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INFLUENCE OF LEAD ABSORPTION ON THE RATIO OF LARGE TO SMALL LYMPHOID CELLS.

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IN investigations of the effects of lead and its compounds on the blood cells, the amount of attention given to the effects on the red cells has been far greater than that given to the effects on the white. Numerous papers have appeared dealing with the red-cell anæmia, poikilocytosis, polychromasia and basophilic stippling. The last-mentioned phenomenon is very readily recognized, and is one which, although it occurs in other conditions than lead poisoning, is still notably characteristic of that malady, at least when present to a considerable degree. The significance of changes occurring in the white cells has been largely overlooked or neglected. That there are significant changes has even been denied.

Possibly as a result of the greater attention given to the red cells there have been considerable differences of opinion as to whether there are any definite changes in the white cells in lead poisoning.

Goadby (1912) stated that in the early stages of lead poisoning, more especially in acute cases, distinct leucocytosis was present, mostly in relation to lymphocytes rather than to polymorphonuclear cells. Large mononuclear leucocytes were greatly increased in number. He further stated that a differential leucocyte count in cases of lead poisoning, which also show the presence of basophilic granules, invariably brings to light an increase in the percentage of lymphocytes and a decrease in the number of polymorphonuclear cells, even when the total leucocyte count is not outside normal limits. In his opinion, while an estimation of the total number of red cells and of white cells was useful, it was not by any means so valuable as the differential leucocyte count and search for basophilic granules. Biondi (1934) states that in mild cases of plumbism there is a tendency towards leucocytosis, although slight, and in more severe cases towards rather pronounced neutrophile leucocytosis. He had observed in certain lead workers mononuclear leucocytosis with morphological changes in the white cells, even in the absence of punctate basophilia. Aub *et alii* (1927) stated that lead was said to produce relative lymphocytosis; but their differential leucocyte counts had not strikingly demonstrated this, the average percentage of mononuclear leucocytes being 26.

Pepper and Farley (1933) stated that there was little effect on the leucocytes in clinical cases.

Bell *et alii* (1925), in considering the effects of lead in the treatment of cancer, found slight leucocytosis with moderate doses of lead and pronounced leucocytosis with large doses. They also occasionally found relative lymphocytosis, and stated that slight relative eosinophilia was fairly common.

Brookfield (1928) found with cancer patients suffering from lead intoxication that the only constant change was a tendency towards an increase in the percentage of monocytes of from three to five.

Gould, Kuhlman and Sheket (1937), in 14 out of 22 cases of cancer treated with lead, found leucocytosis. The differential leucocyte counts showed an absolute increase in the percentage of neutrophile cells (98% of the total in some cases); in the percentage of lymphocytes there was a relative and absolute decrease to as low as 1%. In no case was any lymphocytosis present. In one case there was slight monocytosis of 11%.

McLean (1930), in investigations of the effect of intravenous injection of colloidal lead into rabbits, found immediate leucocytosis due at first to neutrophilia; after eight hours the numbers of lymphocytes and large mononuclear cells began to increase, and in two days reached a higher maximum than the granular cells. Subsequently the white cells diminished in number and the non-granular cells remained in excess of the granular cells. The lymphocytes exhibited a greater response to the lead injections than did the polymorphonuclear leucocytes.

Ferguson and Ferguson (1934) carried out an extensive investigation into the effects of lead on the blood of men exposed to varying lead hazards in ship-breaking. They found that an early effect of lead compounds was an increase in the ratio of large lymphocytes *plus* monocytes to small lymphocytes. As long as the ratio stayed at a moderately high figure clinical symptoms were not encountered, but a fall in the ratio, especially to below 2·0, was an indication of impending plumbism. They found that the increase in the percentage of large mononuclear cells was more closely related to the clinical condition than was the stippled cell count, or the blue line, or the red cell count, or the percentage of hæmoglobin. They considered that for practical control purposes the ratio of large to small lymphoid cells was the most useful test in regard to the prevention of plumbism. They emphasized that their findings might not be applicable to other types of lead industry, where the hazards might be different from those obtaining in ship-breaking; in this industry oxy-acetylene torches were used on ships painted with lead compounds.

Cantarow and Trumper (1944), after referring to a number of authors' conclusions, state that the great majority of observations contradict these specific effects of lead upon the relative proportions of the white blood cells in the peripheral circulation in uncomplicated cases (lead).

Possibly the reason why the findings of Ferguson and Ferguson have not been more generally accepted is that, in so far as the relative numbers of large mononuclear cells and small lymphocytes are concerned, the condition found in cases of lead poisoning is similar to that found in persons who have had no significant exposure to lead or its compounds. Hence it has been too readily assumed that lead has no effect on the lymphocytes, or at any rate no well-defined or characteristic effect which would be of any aid in the study of lead poisoning or of any general hæmatological interest.

The present author (Shiels, 1936, 1937), in investigations covering hundreds of persons engaged in lead mining and smelting *et cetera*, has confirmed the general findings of Ferguson and Ferguson in regard to the ratio mentioned above.

Lane (1949) states that changes in the white cell ratio are not sufficiently constant to be of practical value.

Müller (1938) stated that painters working with white lead were found to have increased lymphocyte counts without other evidence of lead intake or lead poisoning, and pointed out the importance of these observations for factory doctors.

Present Investigations.

The present paper shows the results of further investigations into the effects of lead on the cells of the lymphocytic series. By lead is meant lead or compound of lead other than tetraethyl lead. In a previous paper the author had demonstrated the greater usefulness in the diagnosis and prevention of lead poisoning of the ratio of large lymphocytes *plus* monocytes to small lymphocytes as compared with the stippled cell count. No opportunity then arose of investigating the change in the magnitude of this ratio as exposure to lead of new workers increased, and of relating it to the increasing absorption of lead as indicated by the urinary lead concentrations. An opportunity recently occurred of studying these effects in the employees of a large new storage battery works. The persons who were considered in this part of this investigation were all new to this type of work, and had not previously worked in any trade involving exposure to lead or its compounds. They are called "new" workers.

Also considered are a large number of the employees at Mount Isa Mines, Queensland (large silver lead mines) who were engaged in mining, milling, smelting and assaying of lead ore, and a considerable number of others engaged in the manufacture of lead storage batteries, in lead wiping of motor-car bodies, in the grinding of materials containing lead compounds, in the manufacture of or association with the use of lead arsenate, in plumbing, in the breaking up of old battery plates, in the manufacture of sheet lead and lead pipes *et cetera*, and in the manufacture of collapsible lead tubes. In one case the occupation was known but the source of lead was not.

None of the persons considered was exposed to tetraethyl lead, and none of the conclusions arrived at are to be taken as necessarily applicable in the case of this compound. They may be, but it is not known whether they are or not.

Methods Employed.

The concentrations of lead in the urine were determined in most cases by the method of Taylor (1925) slightly modified by the author (Shiels, 1936). The concentration has been expressed in milligrammes per litre. It has also been the practice in this laboratory for some time to express the concentrations in terms of microgrammes of lead per gramme of solids in the urine; but since this practice has not become common it was thought better to adhere to the more usual method of expression.

The ratio of large lymphocytes and monocytes to small lymphocytes was determined by the method previously described (Shiels, 1936). As large lymphocytes were classified those equal to or greater than 10μ in diameter. As there is some difference in the distribution of the cells according to size at the edges of the film and towards the beginning and end of it, the cells were counted in strips across the film from edge to edge. In some earlier cases this was done in the central portion of the film only; but in the work on the "new" employees the cells were counted across the middle region and across regions near each end. In well-made films, when the film was only one cell thick, it was found that the ratio determined in the middle region agreed well with the ratio obtained from the total counts at all three regions. When only one area was examined at least 50 lymphoid cells were counted, and when the three areas were examined about 30 to 50 cells in each area were counted.

The stain used was Leishman's. With this technique the ratio for non-exposed healthy persons is of the order of 1·0.

Investigation of New Workers.

The proportion of stippled cells, the ratio of large lymphocytes *plus* monocytes to small lymphocytes and the urinary lead excretion were determined at each examination at intervals of approximately six weeks. The stippled cell counts will not be considered in this paper. The first examinations took place after the employees had been working for several weeks in this factory. In the factory a considerable amount of care had been taken to minimize the lead hazard by suction exhaust ventilation and so on; but gradually there was an increase in the atmospheric lead concentrations in a number of places. There was thus, generally speaking, an increase in the total lead exposure, either by an increasing length of exposure to a non-increasing daily hazard, or by exposure to increasing lead concentrations in the atmosphere.

Table I shows the distribution of the values and the average values at the successive examinations. The number of subjects at the second examination was a little smaller than at the first, owing to the fact that some employees had left the works.

This table shows an obvious increase in the percentage of persons having higher values for the urinary lead concentration and for the cell ratio at the second examination. For instance, only five out of 43 persons had a ratio greater than 4·0 at the first examination, whereas at the second examination 30 out of 38 had a ratio greater than 4·0.

Similarly only one out of 43 had a urinary lead concentration greater than 0·15 milligramme per litre at the first examination, whereas at the second 22 out of 39 had a concentration greater than this. That the increases in the ratio and in the urinary lead concentrations between the first and second examinations were not due to chance has been shown by considering the increases in the values for the same individuals at the two examinations and submitting the results to statistical analysis.

TABLE I.

New Workers: Distribution of Values for the Ratio of Large Lymphocytes plus *Monocytes to Small Lymphocytes, and Urinary Lead Concentration.*

Observation.	First Examination.		Second Examination.	
Ratio of Large Lymphocytes *plus* Monocytes to Small Lymphocytes.	Number of Persons.	Percentage in the Class.	Number of Persons.	Percentage in the Class.
0 to 2·0 ..	20	46·5	2	5·3
2·1 to 4·0 ..	18	41·9	6	15·8
4·1 to 6·0 ..	3	7·0	14	36·8
6·1 to 8·0 ..	2	4·7	8	21·1
8·1 to 10·0 ..	—	—	5	13·1
10·1 to 12·0 ..	—	—	2	5·3
12·0 and over ..	—	—	1	2·6
Average value ..	43 persons	2·076	38 persons	6·10
Urinary lead concentration—				
0 to 0·05	10	23·3	5	12·8
0·06 to 0·10	25	58·1	5	12·8
0·11 to 0·15	7	16·3	7	18·0
0·16 to 0·20	1	2·3	9	23·1
0·21 to 0·25	—	—	8	20·5
0·26 to 0·30	—	—	3	7·7
0·31 to 0·35	—	—	1	2·6
0·36 to 0·40	—	—	1	2·6
Average value ..	43 persons	0·078	39 persons	0·167

For 33 individuals the mean increase in the ratio at the second examination was 3·66, the standard deviation was 3·554 and the standard error 0·618. Calculation of the *t* value of Fisher (1928), which is obtained by dividing the mean increase by the standard error, was 5·92. From Fisher's table this value for 32 observations (one less than the number of observations of the increase) corresponds to a *P* value less than 0·001 and is thus highly significant.

TABLE II.

Value Determined.	Number of Determinations of Increase in Value.	Mean Increase.	Standard Error of Mean.	*t*	*P*[1]
Ratio	33	3·66	0·618	5·92	<0·001
Urinary lead concentrations	32	0·085	0·0131	6·48	<0·001

[1] When $n = 30$ and $t = 3·646$, $P = 0·001$.

Similarly, *t* values for the increase in urinary lead concentrations for 32 of the same persons (one subject was not tested at the second examination owing to absence) was 6·48. For 31 observations this corresponds to a *P* value less than 0·001, and again is highly significant.

These results are shown in Table II.

A *P* value of 0·01 corresponding to a certain *t* value means that there is only one chance in a hundred that the value of *t* will be exceeded by chance. If *P* is less than 0·001, there is only one chance in several thousands that a *t* value of 3·646 will be exceeded by chance, whereas the values are 5·92 and 6·48.

Correlation Coefficient.

Further evidence of the influence of lead absorption on the cell ratio is found by determining the correlation coefficient between the ratios and the corresponding urinary lead concentrations found at the two successive examinations.

In a previous paper (Shiels, 1937) it was stated that the correlation coefficient between the cell ratio and the urinary lead concentrations of a number of subjects of lead poisoning who had been exposed to lead hazards for

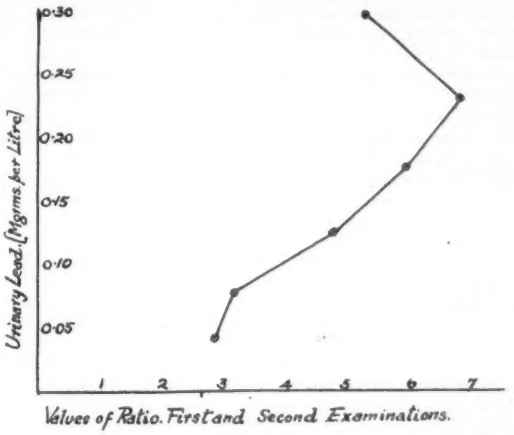

FIGURE I.

many months was 0·71. It has since been found that there was an error in the method of calculating the coefficient, and that there was no direct correlation within the group of lead poisoning cases.

In the case of new workers, however, there is a significant correlation between the two magnitudes.

The blood films were taken and a bottle was supplied at the same time. Urine was collected on that day and on the following morning. The urinary lead concentrations

TABLE III.

Number in Class.	Urinary Lead Concentration.		Cell Ratio: Average Value.
	Range of Values.	Average Value.	
15	0 to 0·05	0·041	2·88
31	0·06 to 0·10	0·078	3·17
14	0·11 to 0·15	0·128	4·77
11	0·16 to 0·20	0·175	5·92
5	0·21 to 0·25	0·228	6·8
6	0·25 and over	0·295	5·26

may be reasonably used as a measure of the blood lead concentration. The urinary lead values ranged up to 0·37 milligramme per litre (see Table I).

This correlation coefficient for 82 pairs of observations was 0·563. From Fisher and Yates's tables this corresponds to a *P* value of 0·001. This means that the two factors considered are closely related. Of course, the presence of lead in the urine could not cause any alteration in the ratio; but the concentration of lead in the urine is closely related to that in the blood. It is realized that sometimes a high blood lead concentration may be accompanied by a low urinary lead concentration and *vice versa*; but generally speaking the two are closely related. It is thus reasonable to regard the lead concentration of the urine as a measure of that of the blood. It is, therefore,

practically certain from the low *P* value that there is a direct causal relationship between the lead in the blood and the cell ratio.

The correlation coefficient between the increase in the individual values of the ratio and the increase in the individual values of the urinary lead concentration at the following examination over the value at the first examination has been determined. The value in this case was 0·4460 for 32 pairs of observations. This corresponds to a *P* value of 0·01. There is thus a significant relationship between the increase in the ratio and the increase in the amount of lead in the system.

The relationship between the ratio and the urinary lead concentration is illustrated in Figure I, in which the average cell ratios of the group having a certain range of urinary lead concentrations are plotted against the average urinary lead concentration for that particular group. The results, which are plotted in the graph (Figure I), are shown in Table III.

The graph in Figure I shows that there is approximately a straight-line relationship between the values up to about 0·20 milligramme per litre.

The increase in the ratio with increasing concentration of lead in the urine does not go on indefinitely. It is known from other evidence discussed in a previous paper

TABLE IV.

Range of Cell Ratio.	Persons with Lead Poisoning.		Same Persons after Recovery.	
	Number.	Average Value.	Number.	Average Value.
0 to 0·5 ..	3	1·28	0	3·95
0·6 to 1·0 ..	15		0	
1·10 to 1·5 ..	16		0	
1·6 to 2·0 ..	15		2	
2·1 to 2·5 ..	—		(value 2)	
2·6 to 3·0 ..	—		12	
3·1 to 3·5 ..	—		5	
3·6 and over ..	—		7	
			23	
Total	49		49	

(Shiels, 1937), and from that of Ferguson and Ferguson (1934) that too much lead in the system acting for too long causes a fall in the ratio. There appears to be some indication of this effect in the last part of the curve in Figure I, but the number of observations of urinary lead concentrations equal to or greater than 0·25 milligramme per litre was small in this series.

Persons With Longer Histories of Exposure.

The Ratio During Lead Poisoning and at Recovery.

Another method of approach is to study the individual cell ratios for a set of persons who are suffering from lead poisoning and for the same subjects after they have recovered and are well. This has been done for 49 subjects. This number includes 28 who were diagnosed as suffering from lead poisoning by the Medical Board at Mount Isa, Queensland, and 21 subjects in Melbourne. These latter subjects were either hospital patients or ambulatory subjects sent to the Industrial Hygiene Division for confirmation of a diagnosis by general practitioners and specialists. There was no doubt that they were suffering from lead poisoning from the clinical condition, the stippled cell counts, the urinary lead concentrations, and other blood tests such as red cell counts or hæmoglobin determinations when these were considered appropriate.

Table IV shows the distribution of the values of the ratio in the two classes.

The average cell ratios of these persons when suffering from lead poisoning was 1·28 and when well 3·95. In regard to the individual increases in the ratio after passing from the "ill" to the "well" condition, the mean increase was 2·67 and the standard error 0·278. The *t* value was 9·60. The corresponding *P* value for such an array of 49 is less than 0·001, showing that the increase in the cell ratio after passing from the "ill" to the "well" condition is highly significant. Occasionally a person exhibiting symptoms will have a ratio greater than 2·0. The persons whose findings are included in Table IV were not specially selected so as to have cell ratios less than 2·0 in the first instance. In the occasional case in which the cell ratio was greater than 2·0 when symptoms were present, the values for the recovered state were not available or other complicating factors were present, such as the presence of simultaneous benzene poisoning, or bismuth therapy in syphilitics.

A somewhat similar method was to compare two different classes of persons, the first being composed of those suffering from severe lead poisoning, and the second of those who were exposed to a lead hazard as indicated by the nature of their work, by stippled cell count, or by significant amounts of lead in the urine, or both, but who were not showing any symptoms of plumbism. All the persons in the latter class worked at Mount Isa lead mine. There were 65 subjects in the first class and 109 in the second. The results are shown in Table V.

TABLE V[1].

Range of Cell Ratios.	Persons with Lead Poisoning.		Persons without Lead Poisoning, but Exposed to Generally Similar Hazard.		
	Number.	Average Value.	Number.	Percentage of Total in Class.	Average Value.
0 to 0·5 ..	8	1·21	0	0	3·72
0·6 to 1·0 ..	23		0	0	
1·1 to 1·5 ..	19		3	2·8	
1·6 to 2·0 ..	9		11	10·1	
2·1 to 2·5 ..	3		17	15·6	
2·6 to 3·0 ..	1		17	15·6	
3·1 to 3·5 ..	1		18	16·5	
3·6 and over ..	1		43	39·4	
Total ..	65		109		

[1] The data in this table formed part of a table appearing in a previous article by the author in this Journal on April 10, 1937.

The average cell ratio for the first class was 1·21 and for the second 3·72.

There is a striking difference in the distribution of the values. Thus, in the class of persons suffering from plumbism there were only six out of 65 with a cell ratio over 2·0 whereas in the state of recovery the percentage with a cell ratio over 2·0 was 87·1.

The *t* value as determined by the method of Fisher (1928, 1936) was 10·51. The corresponding *P* value for 174 observations is less than 0·001. This indicates that there is a highly significant difference between the cell ratios in the two classes.

A much larger array of lead-poisoned individuals and of persons engaged in lead trades and exposed to significant hazards, but not suffering from lead poisoning, has also been considered by combining the observations relative to workers at Mount Isa Mines, those relative to 50 cases of lead poisoning in Melbourne, and those on 156 persons working in lead trades in Victoria but not lead-poisoned.

Observations on 115 lead-poisoning subjects and on 265 persons not suffering from lead poisoning, but exposed to significant hazards, are available for comparison. In a few cases the same person figures in both the "lead-poisoned" and "non-lead-poisoned" condition; but the majority of the 380 observations refer to different subjects.

The results are set out in Tables VI and VII, so that comparison can be made between persons working in a lead mine and others working in a variety of trades (battery workers predominating).

The statistical findings are summarized in Table VIII.

TABLE VI.

Range of Cell Ratios, Lead Poisoning Subjects.

Range of Cell Ratios.	Melbourne.		Mount Isa.		Total, Melbourne and Mount Isa.		
	Number in Group.	Average Value.[1]	Number in Group.	Average Value.[1]	Number in Group.	Percentage of Total in Class.	Average Value.[1]
0 to 0·5	2		8		10	8·7	
0·6 to 1·0	12		23		35	30·4	
1·1 to 1·5	20		19		39	33·9	
1·6 to 2·0	14	1·43	9	1·23	23	20·0	1·32
2·1 to 2·5	1		3		4	3·5	
2·6 to 3·0	—		1		1	0·9	
3·1 to 3·5	—		1		1	0·9	
3·6 and over	1		1		2	1·7	
Total	50		65		115		

[1] Average value for comparison of means, 1·3.

The cases of lead poisoning at Mount Isa were mostly among the miners, with a smaller number from the smelters, mill and assay office workers and general workers on the surface. At the time at which these cases occurred the ore mined was mostly sulphide, very little carbonate being then produced. Some of the subjects had worked in the carbonate zone in the days when the greater production was of carbonate. Some had worked only in the sulphide zone, yet were definitely suffering from plumbism with high urinary lead excretion (up to 0·48 milligramme per litre—one unusual subject had 1·25 milligrammes per litre). The Melbourne subjects worked in a variety of trades, battery workers predominating. There is not much difference in the cell ratios in so far as the total percentages up to and beyond 2·0 are concerned. There is some difference in the distribution of the ratios in the ranges below 2·0, Mount Isa yielding the greater proportion of the lower ratios (up to 1·0) and a lesser proportion of the ratios from 1·1 to 2·0. This was probably due to the fact that the workers at Mount Isa were usually examined within a day or two of leaving their work, whereas in the Melbourne cases considerably longer periods frequently elapsed between the time persons left work and were examined by this Division, and some slight improvement in condition had probably taken place meanwhile. There was one unusually high cell ratio among the Melbourne cases (8·6). If this is excluded, then the average cell ratio for the Melbourne cases is 1·29, compared with 1·23 for the Mount Isa cases.

In regard to the non-poisoned persons, the distributions of the ratios agree closely. Thus for ratios up to 1·5 the percentages of the totals are 4·6 and 2·8 for the Melbourne and Mount Isa cases respectively; for values up to 2·0 they are 12·9 and 12·9 respectively; for ratios up to 3·0 they are 43·6 and 44·1; and for values over 3·0 they are 56·4 and 55·9 respectively.

The average cell ratios were 4·57 for Melbourne cases and 3·76 for Mount Isa cases, the higher average for Melbourne cases being due to more of the higher values in the range above 3·6. There were 11 ratios over 10 in Melbourne and two at Mount Isa. If these are excluded, the averages become 3·77 for Melbourne cases and 3·61 for Mount Isa cases.

A comparison of the distribution of the cell ratios in lead poisoning cases (last column, Table VI) and for persons not suffering from lead poisoning but exposed to lead hazard (last column, Table VII) for these larger numbers shows the same sort of difference as was indicated in Table V.

The mean ratios of these fairly large arrays of 115 persons suffering from lead poisoning and 265 persons exposed to lead hazards but not suffering from lead poisoning have been compared by the method of Fisher previously mentioned. The t value calculated by the previous formula was 10·204.

When $n = 120$, a t value of 3·375 corresponds to a P value of 0·001. Hence for this larger array of 380 observations the t value of 10·204 would have a P value much less than 0·001. There is thus a highly significant difference in the values for the two classes of persons.

In Tables VIIIA and VIIIB, A, B, F and G refer to persons employed at a new battery works, D refers to employees at a large silver lead mine, C refers to 28 employees at a large silver lead mine and 21 in a variety of occupations, and E refers to 174 employees at a silver lead mine and 206 in various lead trades.

Discussion.

That there is a significant increase in the ratio of large lymphocytes *plus* monocytes to small lymphocytes among new workers with increasing time of employment in a

TABLE VII.

Range of Cell Ratios, Persons not Lead Poisoned.

Range of Cell Ratio.	Melbourne.			Mount Isa.			Total, Melbourne and Mount Isa.		
	Number in Group.	Percentage of Total in Class.	Average Value.[1]	Number in Group.	Percentage of Total in Class.	Average Value.[1]	Number in Group.	Percentage of Total in Class.	Average Value.[1]
0 to 0·5	0	0		0	0		0	0	
0·6 to 1·0	2	1·3		0	0		2	0·75	
1·1 to 1·5	5	3·3		3	2·8		8	3·0	
1·6 to 2·0	13	8·3	4·57	11	10·1	3·76	24	9·1	4·24
2·1 to 2·5	25	16·0		17	15·6		42	15·85	
2·6 to 3·0	23	14·7		17	15·6		40	15·09	
3·1 to 3·5	17	10·9		18	16·5		35	13·2	
3·6 and over	71	45·5		43	39·4		114	43·0	
Total	156	100·0		109	100·0		265	99·99	

[1] Average value for comparison of means, 4·2.

TABLE VIIIa.

Tests Considered.	Number of Persons.	Mean Increase.	Standard Error.	Value of t.	Value of P.	Meaning of P Value.
A. Increase in ratio in successive examinations of the same individuals.	33	3·66	0·618	5·92	<0·001	Very highly significant
B. Increase in urinary lead concentration in successive examinations of same individual.	32	0·085	0·013	6·48	<0·001	Very highly significant.
C. Increase in ratio between condition of lead poisoning and recovery for same individual.	49	2·67	0·278	9·60	<0·001	Very highly significant.
D. Comparison of class of lead poisoned persons (65) with class of non-lead-poisoned persons exposed to similar hazard in lead mine (109).	174	2·516 (Difference between means.)	1·537	10·51[1]	<0·001	Very highly significant.
E. Comparison of class of lead-poisoned persons in various trades (115), with class of non-poisoned persons employed in same trades (265).	380	2·9	2·516	10·204[1]	<0·001	Very highly significant.

$$^1t = \frac{\bar{x} - \bar{x}^1}{s}\sqrt{\frac{(n_1+1)(n_2+1)}{n_1+n_2+2}}$$

$n = n_1 + n_2$, where n_1, n_2 are the numbers of persons in the respective groups.

Where $x_1, x_2 \ldots$ are the values in one group and $x_1^1, x_1^2, \ldots$ are the values in the other group and $S(x)$ and $S(x^1)$ the respective sums of the values,

$$\bar{x} = \frac{Sx}{n_1+1}, \quad \bar{x}_2 = \frac{Sx^1}{n_2+1}$$

$$s^2 = \frac{1}{n_1+n_2}\{S(x-\bar{x})^2 + S(x^1-\bar{x}^1)^2\}$$

TABLE VIIIb.

Correlation Coefficient.	Number of Pairs of Observations.	Correlation Coefficient.	Value of P.	Meaning of P.
F. Between ratio and corresponding urinary lead concentration.	82	0·563	<0·001	Very highly significant relationship.
G. Between increase in ratio and increase in urinary lead concentration at successive examinations.	32	0·4460	<0·01	Significant relationship.

trade with lead exposure has been amply demonstrated. There was no evidence of any other factor than the lead exposure to account for this alteration. There were no cases of any kind of sickness among the employees during this period concerned, apart from one or two minor colds or slight attacks of so-called influenza.

It would be reasonable to attribute these increases to the lead absorbed, even in the absence of any determination of lead in the system. However, when there is shown

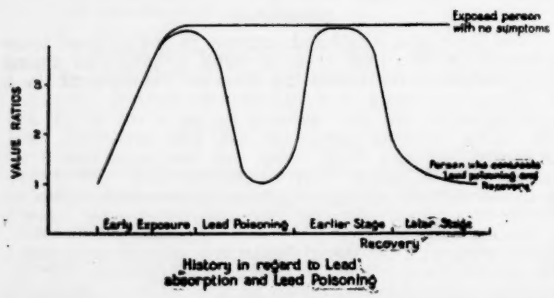

FIGURE II.

to be a significant concomitant increase in the urinary lead excretion and a direct correlation between the cell ratios and the urinary lead concentrations, it becomes practically certain that the former increases were due to the absorption of lead. If the lead exposure is too great, the cell ratio falls again and symptoms appear. On removal of the patient from exposure and/or when he is treated, the ratio again increases. These facts are demonstrated in Table V and Table IV.

In the early stages of recovery, when the patient is symptom-free and feeling well, there may still be sufficient lead in the system to cause the cell ratio to be well above the average for non-exposed persons.

If the patient is examined late in the recovery period and the system is practically free from circulating lead, it will be found that the ratio has fallen to within the range found for persons with no lead exposure. Direct evidence of this has not been adduced in this paper, but experience with large numbers of cases indicates that this is so. It will be discussed in another paper on citrate therapy in lead poisoning.

The sequence of events in regard to the ratio and lead absorption is as shown in Figure II.

Mode of Action of Lead.

At the present time no answer can be given to the question why there is an increase in the cell ratio in the presence of lead. It may be, as has been suggested by various workers, that the lymphocytosis which occurs on exposure to lead is of the nature of a defensive reaction.

The change in the cell ratio may also be part of this defensive reaction, if one assumes that the larger lymphocytes are younger forms which are poured into the blood in increasing numbers to deal with the toxic substance.

On the other hand, it may be that lead in small amounts stimulates the production of monocytes and the larger and possibly younger forms of lymphocytes.

It is known that exposure to radioactive substances and to X rays has a pronounced effect on the lymphocytic cells.

A few observations in this laboratory appear to indicate that the exposure to irradiation to which X-ray technicians are subject causes an increase in the cell ratio, and it is proposed to continue these observations.

Summary.

1. The results of determinations of the ratio of monocytes *plus* large lymphocytes to small lymphocytes in a series of over 400 persons employed in various lead trades have been discussed.

2. Absorption of lead causes an increase in this ratio of monocytes *plus* large lymphocytes to small lymphocytes.

3. In cases of lead poisoning this ratio falls again to within the range of the ratio for non-exposed persons.

4. If the ratio falls to less than 2·0, lead poisoning is imminent, if not already present.

5. Recovery from lead poisoning is accompanied by a rise in the ratio to above 2·0 in the early stages. In the later stages of recovery the ratio again falls to the range of ratios of non-exposed persons.

6. In the cases of new workers there is a direct correlation between the cell ratio and the urinary lead concentrations as exposure increases.

7. The value of the cell ratio is a valuable guide to the prevention and diagnosis of lead poisoning.

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PSYCHIATRY AND EUGENICS.[1]

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EUGENICS as a term was first used by Sir Francis Galton in 1883 to designate the reform movement introduced by him for improving the human race by scientific breeding. In those days many fallacious ideas about ancestry were held, the true mechanism of heredity not then having been discovered. For instance, it was then believed that the upper classes belonged there on account of their superior heredity and that the lower classes had an inferior heredity. Naturally, this attitude was greatly resented by the more democratically minded, and consequently the eugenists, although naturally strongly supported by the aristocracy initially, made little progress. It was not till later, when Mendel's work on heredity was published, that Galton's followers received renewed support in regard to their theory that most of the major ills were influenced by heredity.

It has been stated that there is no sign that any progress in mental capacity has taken place since the days of the ancient Greeks; and further, it has been asserted that the human race is succeeding in producing proportionately fewer persons of eminence than did these same ancient Greeks; briefly, what is meant is that the human race is actually morally and intellectually worse off now than it was 2000 years ago.

In this connexion eugenists have made the following rather sweeping statements.

1. Insanity, feeble-mindedness, epilepsy, pauperism and certain forms of criminality are on the increase—stressing the fact that eugenic measures are the only possible protection society has against further deterioration.

2. Defective people propagate at a greater rate than do the normal members of the population.

3. The conditions under discussion are fundamentally and mainly hereditary.

4. Environment is of much less importance than the germ plasm in the creation of adverse social status, mental disease, criminality and social maladjustment.

The statement that insanity is on the increase is perhaps questionable and is certainly difficult to prove. Statistics dealing with this question are fallacious and are influenced by a number of factors. As a result of modern advancement in medicine longevity of the population is increasing, and there is a greater tendency towards the survival of the so-called unfit. Consequently we are now being faced with the increasing problem of providing for more and more senile subjects shelter and refuge, more often than not in mental hospitals. As the population becomes more urban it becomes more difficult to care for the elderly, the defective and the mentally ill in their own homes. As a result such people are placed in institutions far more often than if the community remained rural. Then again, as mental hospitals improve and can provide more facilities for treatment, and as the stigma formerly attached to such places is removed, there will naturally be a greater tendency for people to have their relatives committed to such hospitals.

It can be definitely stated that as regards mental diseases commitability and recognition are on the increase. It would seem that if statistics as a whole are considered and analysed properly, a curious and somewhat inexplicable contradiction to the usual eugenic statement appears. According to the official year book of the Commonwealth in 1911, the number of insane per 100,000 of population was 98·49, in 1921 the number was 86·49, and in 1928 the number was 79·24.

In New Zealand in 1916 that rate was 97·7, whereas in 1931 it was 89·00.

[1] Presidential address delivered at a meeting of the Australasian Association of Psychiatrists on November 29, 1949, at Hobart.

The committee of the American Neurological Association for investigation of eugenical sterilization reports as follows:

> There is nothing to indicate that mental disease and mental defect are increasing, and from this standpoint there is no evidence of a biological deterioration of the race. Hospital population is increasing, but the alarmist attitude is not justified. No doubt good hospitals and good hospital systems are in themselves good eugenic measures.

As regards the statement made by eugenists that defective people propagate at a greater rate than do normal people, this is denied by the celebrated report of the British Commission in 1937. The results of the commission's investigation were of special value, in that records were taken largely from one social class. Admittedly people of the lowest social class do tend to have larger families than do those of the more privileged classes. The assumption that the former are less fit mentally than the latter is a very debatable point. The larger size of families found in the lower social strata is often due to ignorance and environmental factors rather than to mental deficiency *per se*. It was stated that the average number of children born in families of the feeble-minded does not differ significantly from that of the general population—3·5 as compared with 3·6 of the general population.

Concerning the question of propagation amongst the actual psychotics themselves, it is found that those who are mentally ill to the point of commitment to a mental hospital do not tend to reproduce themselves. Pollock and Furbush quoted 60·9% of schizophrenics, 59·3% of epileptics and 76·4% of mental defectives as remaining unmarried. The committee of the American Neurological Association referred to above made the following statement:

> In those who become mentally sick early the sexual desire is diminished and such people are usually not attracted to the opposite sex. Their queerness is felt, and their inferiority breeds a complex of conditions which prevents their marrying. In those cases who do marry, the birth rate is low and the reputed high fecundity of the mental defective group is a myth. . . .

The eugenists' statement that environment is of much less importance than hereditary influences was violently contested by the opposition—namely, the "environmentalists" and "behaviourists", who insisted that heredity was of little account and that education, hygiene and social improvement alone could bring about a more perfect humanity. Fortunately there is now a tendency for these opposing camps to compromise as it were.

Eugenists now recognize that heredity and environment are complementary forces that should not be considered separately. It is now realized that many defects previously attributed to bad heredity are due primarily to outside influences, and no matter how much heredity is improved it will be of little avail unless an improvement can be made also in environmental conditions. The environmentalists in their turn, faced with the indisputable proof that many abnormal and undesirable characteristics are directly due to or influenced by heredity, must admit that no plan for improving humanity can altogether ignore the importance of improving our genetic make-up.

In order to improve the human race eugenists have put forward a twofold programme in which both environmental and genetic measures are included—to encourage the proportion of the fit and to reduce the proportion of unfit; these are known as "positive" and "negative" eugenics respectively.

As regards positive eugenics, the aim is to increase the rate of multiplication of the great mass of the community, who are well above the average in inborn good qualities. In probably all democratic countries, during at least the past generation, the tendency has been towards a decline in the birth rate. Worse than that, the actual birth rate is unevenly distributed in the various levels, being lowest among those in the upper social and economic levels and highest among those in the lower social and economic levels. One of the principal causes for this disproportion is the growing practice of birth control amongst the so-called more enlightened people, owing no doubt in many cases to the desire to space and regulate the size of their families in accordance with their desire to ensure better opportunities for their children. Not infrequently social ambitions and refusal to accept added responsibilities are responsible for this limitation.

Another cause of disproportion in birth rate is the difference in age at marriage. People whose education and training are prolonged usually marry later in life.

The modern emancipation of woman, with its associated "masculine-protest", has not been at all conducive to large families. Psychological, social and economic forces prevent settling down and inhibit reproduction. Generally speaking, women living in flats or small villas without domestic aid usually think twice before embarking on motherhood.

To counteract these influences, the necessary eugenic measures are social and economic; to list a few: marriage grants, salary increases for married men, satisfactory housing projects with provision of labour-saving devices in the home, larger tax reduction for each child, educational grants, and so on. Possibly even more important than any of these, a change in attitude and outlook on the part of many young married couples would do much in the way of positive eugenics.

Other methods by means of which it is hoped to improve the well-being of the race include the safeguarding of the germ plasm and unborn children from infection, poison and improper nourishment. This is effected by prenatal care and by the beneficial effects which potential parents may have on their germ plasm, and so on their descendants, by means of good mental and physical surroundings.

Better selection or greater wisdom in choice of marriage partners could be another potent weapon were it always practicable. Unfortunately Cupid was never taught genetics. The very formidable list of maladjusted marriages in modern times is not exactly a good recommendation for our present method of free selection. The practice followed by certain races, in which marital partners are selected by the young people's more experienced parents, no doubt with an eye to genetics, could scarcely be any less successful. Maybe there is a great deal to be said for it.

The aim of so-called "negative" eugenics is to decrease the multiplication of all those definitely inferior as regards inborn qualities.

The methods usually recommended for the elimination of the unfit are as follows: segregation, control by contraception, control of marriages in certain cases, and sterilization of the unfit.

Segregation, to a point, is assisted by confining patients in mental hospitals and mental defective colonies. By these means many of the mentally unfit are eliminated very effectively, but its application is somewhat limited. The majority of mental defects in the community are never confined in institutions, and a number of treated psychotics are discharged from mental hospitals as apparently cured and are free to continue propagation of their kind.

The chief arguments put forward in favour of contraception are medical reasons, regulation of intervals between births (a necessity when parents are unable to provide adequately for their children), the prevention of possible transmission of hereditary defect by its absolute use, and its preference to criminal abortion.

It has been suggested in some quarters that compulsory propaganda should be introduced so that information regarding contraceptives would be obtainable by all married women, especially the poor and other unfortunates, and at the same time undue family limitation amongst all superior stocks should be denounced. Although the usual objections raised against these measures are on religious grounds, yet, as has already been pointed out, in actual practice birth control so far has had exactly the opposite effect to that which eugenists desire, being rarely used by the unfit on the one hand and abused by the fit on the other. In spite of the fact that certain authorities have expressed the opinion that this eugenic method is the most powerful agency for racial improvement, there are many

others who regard it as one of the chief factors in racial decline and consider that it will continue to be so under existing circumstances.

Control of marriage could best be brought about by an extension of the work of marriage guidance councils or similar bodies. It should include, at its best, complete physical and mental examination of prospective marriage partners, with a recommendation against marriage, or at least against the bearing of children, should there be on either side any defect with hereditary significance. Such advice, however, could in the present state of our civilization be only advice, and compulsion in carrying it out would not at this time be practicable.

It is, however, the fourth method advocated for the elimination of the unfit—namely, sterilization of the unfit—that has provoked most controversial interest during recent years. The usual arguments directed against this measure are as follows: public opinion; indefensible interference with the liberty of the individual; limited application because seeds of hereditary defect are frequently carried by apparently normal people; increase of the temptation to liberate those individuals whose segregation would, for obvious reasons, be the preferable course to adopt; the supposed increase of promiscuous sexual intercourse; and the possibility of its being applied more harmfully than beneficially from a racial point of view. No doubt, had this method been in operation in the past, many famous and great men, including not a few recognized geniuses, would never have been born. There is a striking list of such people worth far more to society than the cost of maintenance of all State mental hospitals put together. These people would have been lost to the world had sterilization laws been enacted on a compulsory basis a few centuries ago.

On the whole, it would appear from accumulated data over the years that it is reasonable to suppose that high ability has some familial relationship, and that talent tends most frequently to appear in superior groups, even though there are numerous enough exceptions in which high ability springs up in the mass of the race to reassure people who fear that genius and talent may die out because of the differential birth rate. It is also safe to assume that the feeble-minded do not breed geniuses.

The Problem of Sterilization.

It is on the discussion of psychiatric problems of some significance that the question of sterilization is usually focused. These include chiefly the major psychoses (schizophrenia and manic-depressive insanity), congenital mental deficiency, epilepsy, Huntington's chorea and criminality; it is proposed to deal with them here at some length because they have an hereditary significance of some importance.

Schizophrenia.

According to geneticists, schizophrenia can be (but not always is) inherited through multiple recessive genes. It is said there is no known group of plainly environmental circumstances that would produce true schizophrenia without specific predisposition. Most of the near blood relatives of a schizophrenic who share his environment do not develop the mental illness. It must be assumed that they are protected by the absence of a specific genetic predisposition. In support of the genetic ætiology of schizophrenia, statistical evidence would seem to indicate a more frequent occurrence of the disease in families of a known subject of schizophrenia than in the general population. According to Kallman, of New York, the children of a schizophrenic parent are nineteen times more likely to have the illness than the general population. If both parents are schizophrenic, then the incidence is eighty times the average expectancy.

Schizophrenia tends to crop up where there is inbreeding or where marriage between two tainted families occurs. On the other hand, it would seem that it could be avoided by suitable outbreeding. Luxenberger suggests that schizophrenia is a complicated recessive character very widespread in humanity, which may crop up in any social or racial group that limits its marriages by exclusiveness. In brief, possibly the only definite statement that can be made at the present time is that schizophrenia occurs in certain families at a higher incidence than in the general population.

Fortunately, schizophrenia leads to a loss of interest in the opposite sex, so that individuals frequently do not fall in love or, if they are already married, frequently leave their wives or husbands, as the case may be. Thus the disease tends to have a self-sterilizing effect which, coupled with the fact that such patients are usually confined in institutions, often acts as a bar to their reproduction.

In recent years, however, owing to improved psychiatric efficiency in effecting "cures" or remissions, more and more of these people once held in institutions are being discharged from hospital and returned to the community. Thus an added complication is introduced to the problem, and the question is raised of the advisability or not of recommending sterilization prior to discharge.

Manic-Depressive Insanity.

As regards manic-depressive psychosis, the literature appears to be unanimous in declaring its hereditary character. Recent research amongst our own case histories in Tasmania supplies ample proof of this. On the whole the hereditary manifestations are higher than in schizophrenia. The percentage incidence of the disorder amongst children of manic-depressive parents is higher than as if the manic-depressive factor was recessive, and yet it is not high enough to make it a pure dominant. Hoffman suggests a dominant manic-depressive character requiring another factor, perhaps inherited from the other parent, to precipitate the psychosis. It is generally accepted as being an irregular dominant.

Luxenberger states that the probability that children of manic-depressive parents will develop the disease is 33% when one parent alone is affected and 66% when both parents are affected. In practical eugenics he stresses the necessity for exercising great caution, and points to the fact that from marriages of manic-depressive couples children may emerge who have not the genotype for manic-depressive insanity, but only partial characters of it, which may be actually of great positive social and eugenic value. Each case would require careful individual consideration. As a group, manic-depressives are socially productive and extremely useful and successful members of society. It has even been stated that their families produce about four times as many members of the higher social strata as the rest of the population. This may be an exaggeration, of course; but it must be admitted that they certainly contribute more than a few.

Bumke makes the following statement:

> The parents and siblings of manic depressives, though having the hereditary taint, are frequently especially desirable members of human society, clever, efficient, active, full of drive, warm hearted, amiable and sometimes geniuses. . . . I would rather accept in the bargain the diseased manic depressives than give up the healthy individuals of the same hereditary cycle.

One can only say that the problem of the manic-depressive presents complications which will tax the judgement of the wisest board, and so it must be met with conservatism and caution.

Congenital Mental Deficiency.

Congenital mental deficiency would appear to provoke by far the biggest outcry on the part of eugenists in favour of the introduction of sterilization measures. Any of us who have anything whatever to do with mental deficiency cannot help but realize the large numbers of mental defectives in the community and the enormous social problem they create. The frequency with which we meet successive generations of low intelligence would certainly indicate that inheritance must play a tremendous part, although it must not be forgotten that environment also plays a role in the variations of intelligence.

Possibly the majority of low-grade mental defectiveness is directly due to prenatal environmental factors and has little or no connexion with hereditary factors. From a

eugenic standpoint these people are of little consequence, as they are sterile and obviously never marry, if only on account of their obvious mental defect and general helplessness.

The large group comprising the feeble-minded show no definite abnormality and are known only because they make themselves obvious by inadequate social conduct due to their lower mentality. It is in this group that are found the feeble-minded families, and it is because of them that discussions concerning inheritance are usually brought forward. Relevant literature invariably quotes the famous American families—the Nams, Jukes, Kallikaks and Hill-Folk—as being representative of feeble-mindedness in general. It has been pointed out by certain authorities that these families are not representative of the mass of the feeble-minded, and suggested that the majority of the feeble-minded in institutions come from the lower middle classes. Criticism has also been levelled at the technique adopted in the investigations carried out in America on the above-named families, especially the work done by field workers, who passed judgement rather too glibly and surely on people dead three or four generations ago.

The British Commission, in summing up the results of its inquiry on mental deficiency, made the following points.

1. In many cases of mental defect there exists in the family some form of mental abnormality. In the majority of such cases, there is evidence of heredity, but the mode of transmission is at present unknown.
2. Hereditary transmission is known, however, in the case of rare forms of defect.
3. It is possible that some mental defect is determined by a combination of genetic and environmental factors.
4. Some defect is not inherited, but is due to environmental factors.
5. Low grade mental defect is more frequently associated than is high grade defect with environmental factors.
6. High grade mental defect occurs proportionately more frequently in the lowest social stratum. It is possible that selective mating may to a large extent account for this concentration of physical and mental defects. It can be shown that the children of parents one or both of whom are mentally defective, are on the average, below normal, and nearly one-third of such children as survive are likely to be defective and more than two-fifths must be expected to exhibit some degree of mental abnormality.

Some 85·9% of the parents of mental defectives are not defectives themselves, but merely carriers. Excluding cases due to organic disease, the results of the various studies show a larger proportion of inheritance of feeblemindedness than could be expected by any law of chance. Feeblemindedness when hereditary cannot be considered as a simple Mendelian recessive character. It is multifactorial, which means that very little prediction can be made as to the results of sterilization of the feebleminded themselves, nor could there possibly be in the present state of our knowledge, any selection of individuals who themselves are not feebleminded, to sterilize for the prevention of feeble-mindedness.

Certain authorities consider that sterilization should unhesitatingly be recommended in cases of feeble-mindedness, although they do not insist in cases known to be of environmental origin. In the latter instances there may be social as well as biological situations of importance. Even under the most favourable circumstances the majority of those who cannot adequately provide even for themselves find a family of children an overwhelming burden. In these circumstances children may have a minimum opportunity for development.

Epilepsy.

As regards epilepsy, the exact influence of heredity does not yet appear to be fully understood. There now appears to be some doubt concerning the long-held belief that epilepsy is directly inherited. Stein claims that the conclusion that epilepsy is inherited is not justified. Nevertheless the American committee accepted the statement that epilepsy occurred more frequently in the descendants of epileptics than of the non-epileptic, but went on to state that it was transmitted from parent to offspring less frequently than had previously been believed. Apparently only 3·7% of the immediate relatives of epileptics in institutions themselves give a history of epilepsy.

Recent work in connexion with electroencephalographic studies on epileptics should throw considerable light on the subject. In a recent communication Dr. G. Trahair, of Sydney, gives us to understand that recent work on the genetics of epilepsy strongly suggests that cerebral dysrhythmia is inherited as a simple dominant, but its clinical expression as a recessive.

In view of present knowledge it must be admitted that the sterilization of epileptics is considered mainly because of the consequent social situation rather than on account of the biology of the disease. Sterilization is desirable only when the attacks are frequent and if personality changes exist.

Huntington's Chorea.

Although much less prevalent in the community than the four preceding conditions, Huntington's chorea is important in the field of eugenics, especially here in Tasmania, where to our knowledge there have occurred more than 100 cases, all subjects being descended from the one person. It is generally accepted that Huntington's chorea is a heterozygomous dominant. That means that all the children of a victim are confronted with the 50-50 possibility that they too will develop the disease in middle life. Because of the usual age of onset (thirty-five to forty years), a potential victim has usually married and raised a family before he manifests signs and symptoms of the disease. It is estimated that some 46 patients with Huntington's chorea are at present living in this State. On the face of it, and to judge by the large number of children produced by the victims in the past three generations, there would appear to be cause for great alarm for the future. Fortunately, however, many of the affected persons in this generation have remained single, and those who have married have limited their families to a considerable extent. Whether this has occurred because of the individual knowledge of risks of transmitting the disease, or whether it is merely in keeping with the general trend of modern times, which is to limit families almost to the point of extinction, it is difficult to say; possibly both factors are operating. Under these circumstances there seems to be less cause for undue alarm than would have appeared. In other words, the spread of the disease is now at a slower rate than fifty years ago. Nevertheless, to prevent effectively all likelihood that the disease would be transmitted, all children of known victims should be required to be sterilized, even though half of them would be free of the taint and would not pass it on to future generations.

Other Neurological Diseases.

Other neurological diseases of familial nature, such as hereditary optic atrophy and Friedreich's ataxia, are so rare that they scarcely need be considered.

Crime.

The question of sterilization of criminals is an old perennial blazoned forth every now and then by the lay Press. Such references are usually directed towards those guilty of sexual assaults and perversions. Since these crimes are not committed with the idea of procreation, and since procreation is extremely rare as a result of such offences, sterilization would appear to be mainly a form of punishment. In any case it is of no particular value, since it does not interfere with the sexual impulse. Further, any definition of crime must be based on social factors. In point of fact, crime is committed by people of varying types of mentality, personality and genetic constitution. The so-called born criminal must indeed be a rarity, hence the difficulty in determining his genetic make-up.

Most authorities agree that any effort to breed out criminal constitution by any eugenical measure in the present state of our knowledge is not to be recommended. More fruitful approaches to the subject are to be found in social measures of various types, designed chiefly to control environmental factors. Of course it is well recog-

nized that a certain percentage of criminals are mental defectives, and as such should be considered in the light of their deficiency; but even here a good environment can do much.

Discussion.

It is most unfortunate that neither psychiatry nor human genetics can yet be regarded as an exact science. In view of the limitations in our existing knowledge, sterilization of normal people is not justified in order to prevent the appearance in their descendants of such mental diseases as those considered above. The only probable exception would be in cases of apparently normal parents who might eventually become victims of Huntington's chorea.

It is significant that in the United States of America, where in some 27 States sterilization laws with compulsory features were first enacted up to forty years ago, these laws, with rare exceptions, have remained unused. They have been changed, revised, vetoed and amended on a number of occasions. California is the only State where the law has been in force continuously for twenty or more years. Up till 1935 almost 10,000 operations had been performed.

With her usual wise conservatism, Great Britain has so far refrained from passing any legislation in connexion with sterilization, although the matter has been discussed for many years.

Although it can be conceded that compulsory sterilization is unjustified in the present state of knowledge, there is no valid reason why it could not be carried out on a voluntary basis provided that the necessary safeguards were not overlooked. Each case could be considered on its individual merits by an appropriate board consisting of persons who had had special training and experience in the problems involved. The American committee in fact recommended voluntary sterilization in selected cases of certain diseases, with the consent of the patient or those responsible for him. The committee recommended that selective sterilization could be considered in the following diseases (arranged roughly in the order in which sterilization would appear to be indicated): (*a*) Huntington's chorea, hereditary optic atrophy, Friedreich's ataxia and other disabling degenerative diseases recognized as being hereditary; (*b*) mental deficiency of familial type; (*c*) schizophrenia; (*d*) manic-depressive insanity; (*e*) epilepsy. The same committee in its summing up stressed the fact that no great or radical change in the complexion of society could be expected from any such sterilization programme as that recommended above. It was the committee's belief that society need not hurry into a programme caused by fear and propaganda. The problem confronting us is admittedly enormous, but there exists no new social or biological emergency.

This does not mean that the problem should be shelved or that we should adopt a *laissez-faire* attitude in relation to it. To the contrary, it is now time for a prolonged research to be undertaken in which psychiatrists, psychologists, statisticians, geneticists and social workers would collaborate.

In the past investigation of the problems of inheritance, and especially of psychiatric conditions, has been inclined to be haphazard and frequently inexact. However, work already done in this connexion should provide a foundation on which to continue.

Psychiatrists perhaps above all people realize how uncertain and unreliable information gained by history and by the *questionnaire* method often proves to be. Frequently too much significance has been given to the coincidence of conditions; for example, in a case of schizophrenia the tendency has always been to seek for and regard with complacency such information as the fact that an ancestor was alcoholic or "nervy". If further corroboration is not sought, the conclusion may be reached that therefore the incidence of schizophrenia in these conditions is greater than that in the general population. Prejudice and tradition have been responsible for far too many conclusions arrived at in the past.

Tasmania, on account of its small size, its insularity and its relatively static population, should prove to be an ideal State for such investigations. Moreover, practically every familial disease there is occurs in Tasmania.

A certain amount of investigation has already been made, but up to the present time this has been confined to Huntington's chorea, manic-depressive insanity and epilepsy. There is still a great deal to be done, especially in the two last-mentioned conditions. Possibly the two major problems, schizophrenia and mental deficiency, have as yet not received any attention in this regard. Such investigations as would be required in these cases would call for special personnel detailed for this work alone. In these days of lavish spending on Commonwealth social projects, it would not be out of place for the Commonwealth Government to sponsor and finance such work. Information gathered over, say, a period of ten years would be invaluable. Adequate performance of the work would require a central group headed by a full-time director, who would determine the technique of research. It is only by properly organized effort that it is possible for the various problems relating to inheritance, especially of neurological and psychiatric diseases, to be classified, and for the resultant knowledge to become the basis for more appropriate action than is possible at the present time.

Conclusion.

In conclusion, it is important to recognize that many diseases are due to defects of society. Sociology must therefore come to the aid of preventive medicine. Poverty is one of the chief causes of disease, and ignorance one of its first allies. Constructive reform must aim at social justice, education and the teaching of eugenics. It is precisely in those communities where social care is good that we find the evidence of the finest culture and on the whole the best biology, and it is in those communities where social care is poor that the population presents an appalling spectacle of degradation.

THE BRITISH PHARMACOPŒIA FROM THE VIEWPOINT OF THE PHARMACOLOGIST.[1]

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ALTHOUGH most of my discussion will be related to pharmacological consideration of the British Pharmacopœia, I should like to say a few words about the history and background which have given us this invaluable book.

From the earliest times there have been collections of information relating to the treatment, and the ritual and materials used in such treatment, of bodily maladies and disorders. There is, for example, a great collection of texts of this type dating from the seventh century before Christ. These are mainly works dating from the reign of Ashurbanipal, who was king of Assyria about 650 B.C., and even these are largely collected information from the archives of the Babylonian temples. In the form of fragmentary clay tablets in some 10,000 to 12,000 parts these records present a difficult problem in translation, but much has been done by Dr. R. Campbell Thompson, who found that the lists of plants of medical value and their uses contained some hundreds of drugs, of which many can be found surviving in the pharmacopœias of the present day.

The present Pharmacopœia is a far different thing from these records of ritual omens and divination and is primarily characterized by the exacting scientific standards it imposes. There are drugs the pharmacologist has ceased to regard seriously, it is true, but the additions from recent medical research make this proportion minute.

What exactly is the British Pharmacopœia? Primarily it is a book of standards for the materials used in medicine.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on April 27, 1950.

To the pharmacologist, however, it is also a valuable reference text-book of biological assay and posology.

Today we are particularly concerned with the seventh British Pharmacopœia of 1948, the seventh in a series started in 1864, and a pharmacopœia which replaces the last revision of sixteen years earlier.

The most outstanding development is the inclusion of some 155 drugs and preparations which were not included in the 1932 revision or the addenda of the intervening years. If we glance through this list we will see many substances which have been the direct outcome of the intensive pharmacological research of the past twenty years. To name only a few, we have drugs such as mepacrine hydrochloride, desoxycortone acetate (DOCA), dicoumarol, heparin, the sex hormones, picrotoxin, pethidine, succinyl sulphathiazole, thiouracil and methyl thiouracil, phenytoin sodium and trichlorethylene as important additions for which the pharmacologist has shown the basis upon which their efficacy depends.

Many of the drugs of the revision of 1932 have been omitted, so that no longer do we find cinchona bark, lobelia, linum or jalap, but in their place is a large number of synthetic drugs, vaccines and antitoxins, hormones, vitamins or antibiotics.

Modern therapeutics uses more and more the administration of active drugs in tablet form or by injection, and there is a monograph on the preparation of compressed tablets and a special appendix devoted to the preparation of drugs for parenteral injection with details of sterile precautions for dispensing these materials and methods of proving that sterility has been satisfactorily achieved. The recognition of the possible presence of pyrogens in distilled water for injection is shown by the precautions stated in the monograph, where it is required that the water be autoclaved immediately after preparation. Industrial pharmacologists have been aware for years that it is singularly easy to produce pyrogen-free distilled water, but only by such a process can it be adequately stored, and in fact if left uncovered for a few hours prior to sterilization, there is a high degree of probability that it will be actively pyrogenic. In this connexion a biological test is described which is the direct outcome of research in the laboratories of the pharmaceutical industry in Britain. In one such laboratory all batches of material for intravenous injection are tested in this way, and information acquired over the previous seven years on this unusual biological assay, unusual since there is no standard pyrogenic material, has formed the basis of this essential and reliable check.

The methods of sterilization for parenteral injections vary with the material to be sterilized, and here the compilers were faced with the major task of devising techniques suitable for use in the pharmacy instead of the cumbersome and elaborate, but highly efficient, methods used in industry. In the appendix there is a description of sterilization by heating in an autoclave, and it is to be emphasized that the most important point in this technique is to achieve certainty that the medicinal solution was exposed to a temperature of 115° C. for the required length of time. The use of a pressure indicator on the autoclave is not necessarily a guarantee that this has been so. The method of sterilization by heating with a bactericide was introduced in the Fourth Addendum and fills a useful place, since by this means many substances can be sterilized without the considerable destruction which might result from full autoclaving. This method partly takes the place of the sterilization by filtration which would be used for many substances of this nature in large-scale manufacture, and it has been in use for several years now and proves to be suitable and reliable.

For intrathecal, intracisternal and peridural injections no bactericide can be employed, and hence sterilization by filtration is the only method which is available. These injections must be absolutely sterile, since there is no mechanism in the cerebro-spinal fluid capable of dealing with the invading microorganism and it forms a good culture medium for bacteria. As a consequence of the omission of a bactericide such materials must be supplied in single dose ampoules, a practice advisable for all materials to be injected, but in this case essential.

Since most biological substances are given by parenteral injection it will be convenient to consider the status of these drugs in the Pharmacopœia at this juncture.

In the sixth revision there were 21 biological substances listed and a further 14 were added by addenda in the interim years. In the seventh revision, which we are now considering, there are 40 such substances, of which 20 require biological assay, and there is a total of 78 monographs dealing with these materials.

The present century has seen the development of practically all the biological substances we know today, and this fact, together with the existence of stable international standards for these substances, has made their inclusion in the Pharmacopœia essential.

Included among the biologicals are the antitoxins, which have been increased in number to include further members from the gas-gangrene-producing organisms. In 1932 only the antitoxin from *Clostridium perfringens* was official, but now we have *Clostridium œdematiens* and *Clostridium septique* antitoxins as well as the polyvalent form prepared from all three.

The monographs on the antitoxins include the various forms such as formol-toxoid, toxoid-antitoxin mixture, toxoid-antitoxin floccules, and alum-precipitated toxoid. Typhus and yellow fever vaccines are also new to the Pharmacopœia.

The vitamins were poorly represented in the sixth revision, but had been largely incorporated by addenda before 1948.

Insulin has been the subject of much scientific study since 1932, and the average manufactured solid insulin is now more potent than is required by the international standard. This has, of course, no disadvantage, but merely indicates that whereas standards are usually goals of purity in this case this goal is now a poor average. Protamine zinc insulin is included, but owing to the amount of work in progress with globin zinc insulin, the monograph was not completed at the time of publication. A great deal of work has been done by the research staffs of the British manufacturers of insulin, and the tests of potency described in the Appendix represent the carefully propounded result of many years of costly research. One of the most important observations in this connexion is that the tests shall be regarded as acceptable provided that the limits of error can be calculated from the test and fall within a prescribed range. This entirely rules out all methods of testing in which two doses of test and standard material are not given to the test animals simultaneously, and it indicates the degree of care taken in biological assay in Great Britain. There is at present no laboratory in Australia carrying out tests on insulin in this way, and to do so requires a far greater degree of skill and organization than would be imagined from reading this section. While we are considering insulin it is interesting to note that the procedure for preparing the injection of protamine zinc insulin allows the use of "a suitable protamine" in the proportion of 0·75 to 1·25 milligrammes of protamine sulphate for each 100 units of insulin. Since different makers of insulin use different protamine/insulin ratios within this range, the pharmacologist wonders at the significance of mixtures of soluble insulin and protamine zinc insulin frequently reported in the literature on diabetic management.

The old posterior pituitary extract of the 1932 British Pharmacopœia is now supplemented by the addition of injections of the oxytocic and pressor fractions in purified form. This is only to be expected, since Kamm and his co-workers produced these purified fractions over twenty years ago. The biological assay of the oxytocic principle of the posterior pituitary lobe by the guinea-pig uterus method is a difficult and highly technical procedure, and since this has recently been the subject of much research it is hoped that the next pharmacopœial revision will see the use of the chicken depressor method suggested as an alternative.

Heparin is now included, as also is a preparation of liver for use by mouth in pernicious anæmia, and the time should not be very distant when the latter preparation can be reduced to a reliable quantitative basis, when liver preparations can be accurately standardized for hæmato-

poietic activity. The pharmacological background of this type of preparation has been considerably clarified by the work of Karl Folkers in America and Lester Smith and his co-workers in England, who both isolated an active hæmatopoietic principle, vitamin B_{12}, from liver. Developments in this field have been slow because of the difficulties of assaying every fraction and concentrate on patients suffering from pernicious anæmia. The American workers had some advantages in this respect, since Short had developed a microbiological assay using *Lactobacillus lactis* Dorner. There is still doubt upon the singular importance of vitamin B_{12}, for it is possible that there are other factors which are required in addition, and it seems that some preparations of liver produce a greater hæmatopoietic effect than would be accounted for purely on the vitamin B_{12} content. The inclusion, therefore, of a liver extract in the new British Pharmacopœia is timely, and it will probably not be superseded for some years, although better methods of standardization are now available.

Penicillin takes a prominent place in the new British Pharmacopœia and there are eight monographs devoted to preparations of this substance. Two methods are suggested for the assay of penicillin, both relating to a staphylococcal infection. The first, and in my opinion the better, is based on Heatley's cylinder-plate assay, and the second is essentially the serial broth-dilution method of Pope and Stevens. For these assays the standard preparation is a sample of pure penicillin G, and once again the tests should be so designed that they give an integral measure of limits of error.

Penicillin has brought in its train the need for improved vehicles for external application, and there are two creams described for this purpose. They differ only in that one is sterilized, whereas the other contains chlorocresol, and since both are hydrous they need to be freshly prepared and stored at a low temperature after the penicillin has been incorporated. Both these creams owe their useful properties to the incorporation of emulsifying wax, a mixture of higher fatty alcohols and sodium alkyl sulphates, so that they are emulsions of oil in water. Penicillin ointment, in which the base is anhydrous, is very much more stable and should retain its potency for several months at room temperature.

Of the sex hormones described in the new British Pharmacopœia only chorionic gonadotrophin and serum gonadotrophin remain of unknown structure, and consequently these two alone require biological assay.

There are still a few chemical substances for which biological control is required, since their chemical identity is not exactly reproducible from batch to batch. Examples of these are the organic arsenicals, for which there are two main types of test. They need to be tested firstly for undue toxicity and secondly for therapeutic efficacy. Both of these tests are comparisons between standard preparations and the sample under examination, and require that the latter should be not more than 20% more toxic than the standard and have not less than 80% of the efficacy of the standard preparation in experimental infections of *Trypanosoma equiperdum*.

If we consider now a few of the general features of the new Pharmacopœia, we shall notice firstly the change, a gradual and continued trend, now nearly complete, from a formulary to a book of standards, so that each monograph relating to a drug is almost a complete set of standards, tests and assays for that substance. The name of the drug heading the monograph is the official Latin name, and below this is given an official abbreviation for use in prescriptions. The alternative English title is equally official, but it is not necessarily a translation of the Latin.

Capital letters for drugs mentioned in the text and italicized phrases other than the names of plants also imply that these are official and are defined in the book.

Analytically a useful collection of tests for each drug is given, but, of course, this presupposes that no unusual impurities shall be present in the drug which are not normally associated with that substance.

The Pharmacopœia gives a series of doses for the drugs contained therein, and these are intended to form a useful guide to the physician, but are not in any way binding; in fact they are usually rather conservative in quantity, so that frequently larger doses are employed in therapeutics.

The doses are given in the metric and imperial systems, and the two sets of figures are equivalent to the nearest convenient quantity.

The subject of weights and measures is an interesting one, since the metric system is employed throughout the Pharmacopœia. The metric system is the first system of weights and measures which the physician encounters in his student days; it is the system of the scientist and I doubt whether a set of apothecaries' weights could be found in an industrial pharmaceutical laboratory. The Pharmacopœia recommends the discontinuance of the use of the archaic ounce and drachm symbols and suggests that solids be prescribed in ounces (oz.) and grains if the imperial system is used.

Objection has been raised to the possible confusion of the decimal place in using the metric system, but this matter was fully explored some years ago by Finnemore and Large and should present no problem.

As so many new drugs are appearing on the market, almost always in metric dosage, the time has surely come to abandon older systems and to bring our therapeutics into line with the basic sciences.

The Pharmacopœia uses names for drugs which are nonproprietary, so that "Pentothal", for example, is thiopentone sodium, and the General Medical Council has recognized certain approved names recommended by the Pharmacopœia Commission, so that drugs not yet included in the Pharmacopœia should be provided with names which may be used by any manufacturer and which will become official if the drugs are eventually pharmacopœial. This is a very praiseworthy effort to do something to reduce to order the bewildering array of names facing the pharmacist and physician. For it to be a success requires the support of the pharmaceutical industry, and since the present state of affairs is universally deplored surely this suggestion will not pass unheeded.

Progress in pharmacological research is no rapid and so virile that the Pharmacopœia can never be really up to date; already there are many new drugs which probably warrant inclusion, and a little more of the dead wood could probably be pruned away. The pharmacologist would suggest that the older galenicals of squill and strophanthus might be relegated to antiquity, perhaps to be joined by diamorphine, aconite and amidopyrine. It cannot be very long before places are found in the Pharmacopœia for gastric antacids, the new synthetic analgesics, dimercaprol, dimethylphthalate and the antihistamines, which have stood the tests of modern exacting therapeutic demands.

PNEUMONOKONIOSIS IN QUEENSLAND FOUNDRIES.

By DOUGLAS GORDON,
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BETWEEN December, 1946, and December, 1948, all known Queensland foundries—76 in number—were visited. These are scattered along the coast and adjoining hinterland from Mossman, 1090 miles north of Brisbane, to Dalby, 153 miles west of the capital. Table I gives the details of this distribution.

TYPES OF FOUNDRIES.

Table II indicates the number of foundries engaged in different types of casting; three of the "combined" group (that is, foundries casting more than one type of metal) engage in steel casting; in one of these, however, the steel annexe is so small that the amount of work done is negligible. In effect, therefore, three shops do steel casting, and one of these has not been in operation sufficiently long to produce any cases of silicosis. Two foundries, therefore, have produced all cases occurring in steel workers.

SIZE OF FOUNDRIES.

Figure I shows the distribution of all foundries in Queensland and Figure II the distribution of all foundry workers, according to size of foundry expressed in terms of the number of employees. By way of comparison somewhat similar data extracted from Greenburg's survey (1938) of foundries in New York State are also recorded in these figures. There is, however, a difference. The New York figures include pattern-makers and clerical and administrative staff, the Queensland figures actual foundry workers only. It will be noted that though there are only a few large foundries in Queensland, these employ an appreciable percentage of the total labour force.

TABLE I.

Place.	Number of Foundries.	Number of Employees.
Brisbane	46	589
Ipswich	4	143
Toowoomba	3	122
Maryborough	5	104
Bundaberg	1	31
Mackay	4	29
Ayr	1	7
Dalby	1	12
Townsville	1	9
Innisfail	2	9
Cairns	1	5
Rockhampton	3	28
Babinda	1	3
South Johnstone	1	3
Mossman	1	1
Mount Morgan	1	23
	76	1118

METHOD OF SURVEY.

The survey was carried out under three different aspects —environmental, medical and miscellaneous.

Environmental Considerations.

A careful inspection was made of all processes in all foundries.

In approximately 20% of foundries various other investigations were undertaken, such as the following: (*a*) Dust counting was undertaken with Owen's dust sampler (Jacobs, 1946; Badham *et alii*, 1927). In most situations which were tested samples were taken at five-minute intervals for one hour. (*b*) Samples of airborne dust were taken for chemical analysis by means of a Greenburg-Smith impinger (Jacobs, 1946; Badham *et alii*, 1927; United States Public Health Service, 1925). That portion of the sample made up of dust particles below 10μ in diameter was analysed for free silica (Badham *et alii*, 1936). (*c*) Samples of "parting" and "facing" powders were analysed for "free silica". (*d*) Atmospheric conditions were measured: wet and dry bulb temperatures, radiant heat (globe thermometer) and air movement (silvered Kata thermometer, 150° to 145° F.) were all considered (Badham *et alii*, 1936; Bedford, 1946; Bedford, 1948).

TABLE II.

Type of Metal Cast.	Queensland.		New York State (All Foundries).	
	Number.	Percentage.	Number.	Percentage.
Iron	22	29·0	129	41·5
Steel	1	2·6	10	3·2
Non-ferrous	19	25·0	109	35·0
Combined	34	43·4	63	20·3
	76	100·0	311	100·0

Medical Examination.

The medical examination comprised the following: (i) An occupational history, clinical history and clinical examination, with special reference to the respiratory system and the cardio-vascular system. Other investigations were made when symptoms or signs, or both, suggested their necessity. (ii) A full-size X-ray film of the chest was taken. In the metropolitan area films were taken by the Brisbane General Hospital radiological department, in country centres at the nearest hospital with a suitable X-ray plant. With such a variety of sources it is not pretended that the films were taken with a standard technique or that they were of uniform quality. However, with some percentage of "retakes" films of "readable" standard were obtained (Nisbet, 1948).

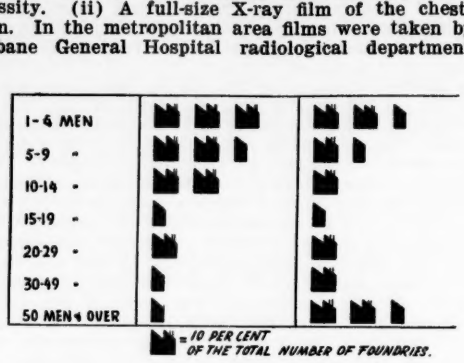

FIGURE I.

Percentage distribution of foundries by number of employees: first column, number of employees per foundry; second column, percentage of total, Queensland; third column, number of foundries, New York State.

Miscellaneous Investigations.

Investigations were made in passing into the following: lighting, metal fume fever, chronic manganese poisoning, noise, vibratory syndrome, *et cetera*.

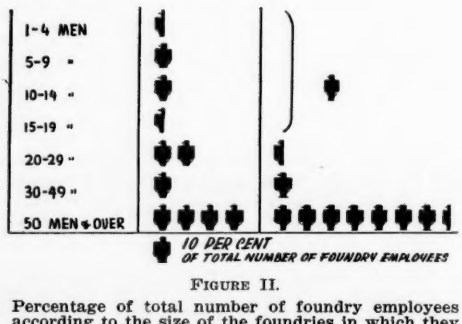

FIGURE II.

Percentage of total number of foundry employees according to the size of the foundries in which they work: first column, number of employees per foundry; second column, percentage of total foundry employees, Queensland; third column, percentage of total foundry employees, New York State.

CLASSES OF EMPLOYEES EXAMINED.

Dr. E. J. Reye of this department had in 1944 conducted a "pilot" survey of 100 foundry workers. From experience so gained he had decided that no useful purpose would be served by radiologically examining moulders with less than fourteen years' trade experience or foundry dressers with less than four. In the field in this survey, however, it was difficult to differentiate between dressers and other iron workers (for often the jobs were interchangeable), and eventually all foundry workers other than moulders were eligible if they had had four years or more of foundry employment. Obviously, however, some of these, such

as moulders' assistants, run no more risk than does the average moulder who was accepted only after fourteen years' trade experience.

These criteria of acceptance being used, 388 men out of 1118 foundry employees were eligible, and of these 359 agreed to cooperate. Other foundry men (though not eligible) who were particularly anxious to be examined were also radiologically examined. These are not included in the survey. No one in this small group was considered "positive" or "doubtful". Table III gives particulars of the age groups of the men who took part in the survey.

The numbers of men radiologically examined, by occupation, are as follows: 203 moulders, 67 dressers, 42 moulders' assistants and 47 other foundry iron workers.

Figure III shows the percentage of the total number in each group in terms of years of trade exposure.

An attempt was originally made to divide the moulders into various categories—iron, brass, steel, *et cetera*—but in practice this was found to be futile, for under Queensland conditions it was discovered that most men had spent a number of years in more than one type of moulding. There had been a similar interchange between "jobbing" and "machine" moulding in many cases. However, the impression was formed that in this State there was far less machine moulding than is usual in the foundry industry in more highly industrialized and mechanized countries. This dearth greatly reduces the risk of silicosis as far as moulders are concerned. Similarly foundry ironworkers' jobs within the industry were often changed, and difficulty was sometimes experienced in fitting men into definite categories—to which the comparatively large number under the heading "Miscellaneous Foundry Ironworkers" testifies. Ironworkers require little or no skill, and in these times of plentiful employment there is a large labour turnover in these jobs, particularly in the city foundries. In some of the latter, few or no ironworkers had the four years' trade experience requisite for the purposes of this survey. This again is a factor which tends to reduce the numbers of men who actually contract pneumonokoniosis.

TABLE III.
Age Groups of the Men Examined and Radiologically Examined.

Age (Years).	Total.	Percentage of Total.
Under 21	1	0·3
21 to 30	44	12·3
31 to 40	113	31·3
41 to 50	88	24·6
51 to 60	86	24·0
Over 60	27	7·5
	359	100·0

RESULTS.

Clinical Findings.

Reports on chest films were made by either Dr. C. Uhr or Dr. K. Uhd, of the Brisbane General Hospital X-ray department. In the great majority of cases occupational data and radiological signs are the main considerations influencing the diagnosis. When equivocal or contradictory opinions resulted from the films, the final decision of necessity became a matter of clinical opinion after consideration of all factors—clinical, occupational and radiological. Originally a number of subjects were placed in the "doubtful" class; but in the three years that have elapsed since the survey commenced most of these men have been radiologically examined again on several occasions and their medical and work records studied from time to time. From this a definite decision has been reached in all cases except those appearing in Table V. "Follow-ups" have, of course, been carried out on the men thought "positive", so that the results as they now appear in Tables IV and V are final opinions expressed usually after several examinations.

Of these 13 subjects giving positive findings, one (Case 5) obviously suffered his exposure in another State. Causation in Case 4 is doubtful, owing to an early history of coal mining. Of the remaining 11 subjects, four were dressers (Cases 1, 6, 10 and 12), three of whom had used pneumatic hammers, and two of these pneumatic hammer workmen were steel dressers. This is the more remarkable since both pneumatic hammers and steel dresssers are few in number in the industry in Queensland. Four can be classed as furnacemen (Cases 2, 3, 7 and 13), and of these two were steel furnacemen. One subject (Case 8) is a machine moulder. Another (Case 11) had a very mixed history, and still another (Case 9) is a jobbing moulder who required fifty years' exposure to show even early signs of silicosis.

Within the last few years the steel foundry which produced Cases 6, 7 and 13 had had four other steel dressers and one furnaceman diagnosed as silicotics prior to this survey. As well as these, another man repairing furnaces in the smithing section also received compensation for silicosis. At any one period there are usually only about half a dozen steel dressers employed in this foundry.

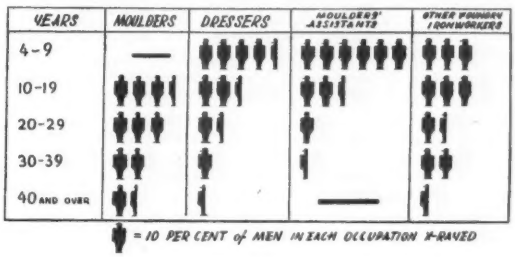

FIGURE III.
Years of trade exposure by occupation of those radiologically examined, and approximate percentages of total number examined in each occupation.

These results confirm the well-known fact that steel foundries present a much greater hazard than foundries doing other types of casting (Greenburg, 1938; Ministry of Labour and National Service, 1944 and 1947), and that furnacemen and dressers are particularly exposed to dust hazards (Ross *et alii*, 1943). There is a suggestion that large foundries under Queensland conditions are more hazardous than small foundries. This is common sense, for in large foundries one man is kept on one job all the time, there are more machines and more men making dust in close proximity to each other, and the smaller buildings of the small foundries as a rule are more likely to be better ventilated by the prevailing breezes. There is also a suggestion that even where the parting powders in the past have been siliceous the jobbing moulder has run very little risk, and in the future with the advent of non-siliceous parting powders the exposure of all types of moulders should be minimal. In "dressing" shops and furnace chipping work of all kinds, however, there are indications that dust suppression measures are most necessary.

Of the 359 subjects radiologically examined, those aged sixty years and over constituted only 7·5% of the total; yet in the eleven "positive" cases definitely produced by Queensland foundry conditions, four of the subjects were in this age group. Similarly, while only 21 dressers with over twenty years' trade experience could be found, this group produced four out of eleven "positive" cases. In addition, of the group of furnacemen numbering in all only 33 covering all periods of trade exposure, there were four "positive" cases. In view of this the short stay in the foundry industry of many dressers and furnacemen may be a blessing in disguise.

Of the eight doubtful cases, it is rather unlikely that in more than one or two the condition would progress to frank silicosis. It was found that observation over a

TABLE IV.
"Positive" Subjects.

Case Number.	Subject's Age. (Years.)	Occupational History Prior to Diagnosis.	Disability.	Present Status.	Classification by X-ray Examination and Prognosis.
1	58	Steel and iron dresser, 20 years in a large foundry. Poor conditions. Pneumatic hammers used.	Slight shortness of breath.	Still dressing. Conditions improved.	Early silicosis Stage I. Outlook moderately good.
2	58	Iron furnaceman in a large foundry. Daily chipping slag; 15 years' exposure.	Light work. Cough ++. Breathlessness ++.	Receiving compensation.	Early silicosis Stage I. Poor outlook.
3	61	Iron moulder doing part-time furnace chipping for 40 years in moderate-sized shop. From medical viewpoint can be classed as iron furnaceman.	Very little.	Foreman moulder. Advised to give up furnace work.	Early silicosis Stage I. Good outlook.
4	57	Iron dresser 9½ years. Conditions reasonable. Coal miner for one year as a lad in a dusty pit (possible cause). Foundry of moderate size.	Nil.	Blacksmith's assistant.	Early silicosis Stage I. Clinically, outlook good.
5	34	Iron dresser for seven years in a very large foundry in another State. One year as moulder in Queensland.	Nil.	Moulding under reasonable conditions.	Early silicosis Stage I. Outlook difficult to state.
6	46	Steel dresser 28 years, large foundry 20 years of this on pneumatic hammers.	Light work. Cough +++. Breathlessness ++.	Compensation.	Silico-tuberculosis radiologically evident, not proved bacteriologically. Outlook poor.
7	51	Steel furnaceman 15 years in a large foundry.	Light work. Disability mainly from *angina pectoris*.	Doing light odd jobs around foundry.	Silicosis Stage II. (?) Silico-tuberculosis. Clinically and bacteriologically inactive.
8	46	Machine moulder—iron, large shop for 32 years.	Nil.	Moulding in good conditions.	Early silicosis Stage I. Clinically outlook good.
9	67	Jobbing iron moulder in large foundry 50 years.	Very slight.	Retired.	Silicosis Stage I. Should not greatly affect expectation of life.
10	65	Iron dresser in large foundry 25 years. Portable grinder and pneumatic chisel.	Unfit for work.	Receiving compensation.	Silicosis Stage II. Poor outlook.
11	58	Mixed history in large foundry; 12 years machine moulding; 15 years preparing sand.	Nil.	Pug mill hand under reasonable conditions.	Silicosis Stage I. Outlook moderately good.
12	62	Iron dresser in large shop for 30 years. At present in a small shop.	Light work only.	Refuses to give up "dressing" under poor conditions.	Silicosis Stage I. Moderately good outlook clinically.
13	47	Steel furnaceman.	Very slight.	Doing odd jobs in foundry. No hazard.	Silicosis Stage I. Clinically probably good.

period of time usually resulted in the great majority of "doubtful" lesions becoming "negative", probably owing to the general tendency in medicine "to play safe".

In this survey no cases of simple uncomplicated tuberculosis of "clinical significance" were found. There was the usual proportion of reports figuring "apical scarring", "old calcified glands", "apical markings—probably inactive" *et cetera*. None of these on investigation suggested recent or active lesions.

Greenburg's figures (1938) are not quite comparable to those of our survey. In his investigation all employees in a representative group of foundries—approximately one-third of the total number—were examined, irrespective of length of trade exposure. In Queensland length of trade exposure was the measure whereby examinees were regarded as eligible. State of New York figures were as follows: foundry workers examined, 4066; silicotics, 110 (15 of these had tuberculosis as well); subjects with fibrosis (that is, "accentuation of normal markings of the lung"), 185; men with "clinically significant tuberculosis" without a silicotic basis, 27. It must be borne in mind, however, that these 4066 are more analogous to our total foundry population (1118) than to the actual number of Queensland foundrymen examined (359).

At the end of 1944 Dr. E. J. Reye had undertaken a "pilot" survey of a hundred foundrymen taken mainly from the larger metropolitan foundries. From this, five "positive" and ten "doubtful" cases were recorded. An effort was made in 1948 to find out the present status of these patients. In the five "positive" cases, one subject had died after rupture of a duodenal ulcer, and a post-mortem examination revealed silicosis. Two other subjects were regarded by the present reviewers as silicotics, but both are elderly, are reasonably well and are still working. Two others—with all due humility—were considered at the most "doubtful". Both are well and still working. Of the "doubtful" group, one subject is now considered a silicotic, six were considered—with wisdom brought by the passage of time—free from silicosis, and of the three who did not report for X-ray examination there is hearsay evidence suggesting that they are well and working.

Environmental Studies.

Atmospheric Dust.

From the mass of data collected in some 15 of the foundries out of the total number visited, very little that was original arose. Most of the findings confirmed work done more completely by other investigators (Ross *et alii*, 1943). The free silica content of air-borne foundry dust of dangerous particle size usually varied between 15% and 20% by chemical analysis. There was some suggestion of correlation between the clinical and environmental findings, but owing to the fact that the "dust" survey did not include all foundries or even a fairly representative sample of them, any forthright conclusions may prove hazardous. Working places which in my opinion seemed to show an appreciable amount of dust were tested, and in practice this meant that in the main only the larger or more confined foundries were so investigated. As instances of this suggestion of correlation the following facts are mentioned:

1. Two out of three steel moulding floors gave hazardous counts, whereas only one out of 15 situations in iron moulding—and that was in machine moulding—gave a high count.

2. Dust in dangerous quantity was found in one out of two situations where pneumatic hammers were being used, but in one place only out of ten grinding operations tested. In the latter place five grinders were in operation together in a somewhat confined situation. Such a number of grinders in the one shop is rather unusual in this State.

3. Blasting cabinets are not usually found in Queensland foundries. Of the three seen one was tested, and owing to defective "aprons" the counts recorded were dangerous.

4. Rumblers are not common but are usually found in the larger establishments. Two were tested, and both returned dust counts indicating a border-line hazard in the light of our present imperfect knowledge.

On the other hand, of five foundries where hand tools were being used to dress castings, a dangerously high count was returned in one only.

The impression was formed that in most "shops" in Queensland "knocking-out" is not particularly hazardous.

TABLE V
Cases Considered of Doubtful Diagnosis.

Case Number.	Subject's Age. (Years.)	Occupational History.	Degree of Disability.	Present Activities.	X-Ray Findings.
14	52	Jobbing iron moulder; 30 odd years in large stove foundries.	Nil.	Moulding.	Indefinite, but progression shown in X-ray films.
15	45	Brass moulder—jobbing in the one shop (moderate size) for 30 years. Was classed as a "positive" in 1944 survey, but X-ray finding now is not regarded as "positive".	Nil.	Moulding.	Indefinite.
16	61	Iron dresser, 16 years in a large shop. No pneumatic tools.	Nil.	Brass dressing.	Indefinite. No response to request for further X-ray examination.
17	60	Brass moulder for 46 years—nine of these in Scotland, 37 in Australian brass shops of average size.	Nil.	Brass moulding.	Doubtful silicosis.
18	59	Iron moulder—jobbing and plate; 47 years mainly in stove foundries of moderate and large size; 10 of these in Scotland.	Nil.	Iron moulding.	Indefinite.
19	44	Steel moulder—jobbing—in moderate-sized steel shop for 25 years.	Nil.	Steel moulding on probation.	Doubtful.
20	40	Brass moulder 24 years in small brass shop.	Nil.	Brass moulding.	Probably clear. Did not respond to request for further X-ray examinations.
21	58	Iron moulder—jobbing for about 40 years in large and moderate sized shops; 14 years in United Kingdom.	Slight shortness of breath.	Moulding.	Indefinite.

This has not been the impression of other investigators (Ministry of Labour and National Service, 1944 and 1947; Ross *et alii*, 1943; Renes, 1949). In most foundries here the operations are so small and the shops so open that the prevailing breeze quickly clears the atmosphere. Only four of the larger foundries were tested, and in only one, which was somewhat confined, were hazardous counts obtained. Wider sampling may have produced different results.

Two opportunities arose for testing furnace chipping, and I can find in the literature only one reference to dust counts taken at this particular process (Renes, 1949). The tests taken here gave 239 particles of dangerous size per millilitre of air and 368 particles of dangerous size per millilitre of air. The one sample taken for chemical analysis gave a result of 45% free silica. These readings, particularly the last-mentioned figure, require confirmation.

Parting Powders.

Parting powders are referred to by Stern (1946), by Ross *et alii* (1943) and by Renes (1949).

At the time of the present investigation the steel foundries were still using highly siliceous powders—silica flour and silica sprays—in moulding. However, since the survey, experiments with a non-siliceous material—"Zircolox"—have been successful and it is hoped that the use of dangerous powders in the steel industry will cease. In other types of moulding a few shops still continued to use brick dust—a hazardous substance. There is no necessity for this, and the practice should be discontinued. Practically all foundries at times use some form of natural sand, containing high amounts of free silica, as parting powders. The latter, however, are not ground, the particle size is large and there is no great tendency for these particles to remain suspended in the air. Under local conditions, at any rate, it is not considered that these types of powders are dangerous.

Miscellaneous Inquiries.

As in foundries the world over, lighting was in many instances poor, owing in part to the smoke-blackened buildings and to the uniformly dark nature of the moulding sand. When it is considered that some processes in jobbing moulding involve fine detail in the use of dark material without any help from colour contrast, this defect is to be deprecated.

No instances of "brass founders' ague" were recorded—probably again owing to the open nature of the shops and the smallness of the industry. One steel dresser suffered from the vibratory syndrome due to the use of pneumatic hammers (Agate *et alii*, 1947). Tests for manganese and a search for cases of chronic manganese poisoning in the steel foundries using manganese were entirely unsuccessful.

DISCUSSION.

Foundry Surveys.

Though it is thought that this is the first complete survey of a foundry industry for pneumonokoniosis in Australia (Ross *et alii*, 1943), plentiful references to such surveys in other countries can be found in the literature. Ross and Shaw in 1943 referred to 15 such surveys over the previous twenty years, and I have seen references to several since then (Vigliani *et alii*, 1948; Renes, 1949; Davis, 1938). The cases of pneumonokoniosis found in these investigations range from slightly over 1% to approximately 70%, and these varying results have greatly perplexed interested but non-medical readers. The explanation probably lies in the type of foundry population surveyed (Ross *et alii*, 1943). When the numbers have been few or only certain foundries have been examined, or when only aged men or men with symptoms have been surveyed, then the results tend to be high. Greenburg's survey on foundries in the State of New York—the principal results of which have been given elsewhere in this article—is one of the most thorough and authoritative examinations of the industry. When surveys are made sufficiently large and completely representative of all foundries in a State or country, the percentage of silicotics to the total foundry "working" population would probably range between 1·5% and 10%, most results being under 5%. Queensland results are in the latter range.

Opinions in the industry vary. Some emphatically deny that a dust hazard exists in foundries, others just as strenuously assert that one-third or more of all foundrymen will die of silicosis (Greenburg, 1938). The truth is that there is a silicosis risk in foundries, but compared with certain other dusty trades—sandstone working, granite cutting, *et cetera*—the risk is mild (Greenburg, 1938). I have formed the impression as far as Queensland is concerned that this risk is not spread uniformly throughout the industry; many "shops" in effect have little risk, while a few foundries have quite bad hazards. The mere statement, therefore, that a man is a foundryman does not necessarily mean that he has been exposed to dangerous concentrations of highly siliceous dust (Ross *et alii*, 1943; Renes, 1949).

Diagnosis.

Diagnosis in a pneumonokoniosis survey is difficult, and presents a different problem from that of the elderly patient complaining of symptoms and perhaps providing a chest film already replete with "snow-storm" markings, or conglomerate shadows, or both. In surveys, most of the subjects are symptomless, and radiological changes—if any—are early. The exact point at which "increased linear fibrosis" found in a percentage of the ordinary aged population as well as in foundrymen becomes "nodular fibrosis", or in the case of coal-miners "reticulation", will always be a matter of individual opinion (*Nisbet, 1948*).

For those of us inclined to be dogmatic, a salutary lesson in humility is to be found in two recent articles. Fletcher and Oldham (1949) state that ten medical practitioners —mostly well versed in the radiology of tuberculosis and pneumonokoniosis—twice reported on a set of 102 films in which early signs of pneumonokoniosis were alleged to be present. In addition two experienced consultant radiologists issued reports on the same films. The opinions of observers, including the radiologists, differed to a remarkable degree, and to a lesser extent the opinions of observers were different on the two occasions on which they read the same films. Nine of the patients had three different films taken on the same day—hard, medium and soft. These authors comment as follows:

> The effect of technique is striking, especially in the cases in standard category 3 near the border-line of certification. Had case 12, for instance, applied to a panel made up of observers in the experiment he would have had a fourfold greater chance of certification if he had applied with his soft film than if he had applied with his hard film.

In somewhat similar vein is a report by Birkelo *et alii* (1947), in which five experienced readers were concerned with estimating the reliability of different types of chest films—14-inch by 17-inch films, microfilms *et cetera*—for diagnosing tuberculosis. When they were limited to one technique "tremendous variation exists among different readers", and "considerable differences are noted also when the same individual reads the same set of films a second time". Alas and alack for the fallibility of man!

In view of the foregoing and of some of my own experiences, I would not be at all surprised if a different set of medical men viewing a certain percentage of the films which formed this survey recorded in many instances entirely different results from those recorded here. While "divine doubt" is most necessary in this work, unfortunately in most cases such as these, industrialists, employees and insurance companies alike require a plain, definite answer. In the majority of cases (made up of the large obviously "negative" group and the small obviously "positive" group, other diseases simulating the radiological signs of silicosis having been excluded (Hamlin, 1948), the radiologist can be so helpful that there comes a tendency to accept radiography as the sole criterion of diagnosis in all cases. However, with that class of patient whose films show early equivocal or doubtful signs, reliance on the most favoured radiologist's report alone sometimes produces "positive" diagnoses which other factors render improbable. In the suspected early case in which the film causes disagreement among experienced observers, the probabilities presented by the clinical and occupational histories become most important. No one can make a certain diagnosis in such cases; and the final opinion must be based on probability, and practitioners experienced in general medicine and occupational hazards—rather than radiologists—should be the ones to weigh carefully these probabilities. For example, two foundrymen may each present "doubtful" films; one may have been a jobbing moulder exposed to a comparatively safe atmosphere in a good moulding "shop"; the other may have worked in a very dusty dressing "shop" which had already produced other definite cases of silicosis. The probabilities in these two cases are obviously different. In practice it is often the non-medical observer who is subconsciously aware of these differences. When a "positive" diagnosis is made in cases such as the first mentioned, employers or fellow employees will often exclaim: "How can old Bill Smith be dusted? I've known him all his working life and he never worked at any job which was dusty." These are observations based on common sense, and a common-sense estimation of the amount of industrial exposure that has really been experienced—often quite distinct from the alleged experience—sometimes throws a deal of light on an otherwise obscure diagnostic problem. Industrial diseases do not occur unless there has been adequate industrial exposure.

Prognosis.

The advice to give a patient considered to be a silicotic is at times a problem, for on the one hand it is a "most cruel kindness" to commit a man capable of work to the unhappiness, idleness and financial stringency of pensions (Fletcher, 1948), and on the other it is catastrophic to hasten his death or unduly expose others in industry to infection.

By and large, most silicotics these days, when first the condition is diagnosed, have years of useful work in front of them (Luongo, 1947; Murray *et alii*, 1946; Gardner, 1945-1946). In spite of much research and many complicated gadgets there is still no sure practical method of estimating individual disability due to pneumonokoniosis (Caplan, 1947; *British Medical Journal*, 1948; Fletcher, 1948*b*). Common sense and clinical acumen are still probably as good guides as any. Unfortunately, in individual cases radiological signs and disability sometimes bear little if any relation to each other (Garrad, 1949; Hart *et alii*, 1942). In view of our present lack of knowledge, it has always been a mystery to me how disability due to pneumonokoniosis can be assessed in highly exact percentages. Presumably medicine is here bowing to legal necessity. It goes without saying that the actively tuberculous should not continue to work. In practice it is at times difficult to state dogmatically just when a patient's pneumonokoniosis has become complicated by tuberculosis. Many lesions, in spite of most rigorous search, fail to reveal themselves as bacteriologically active (Sander, 1949; Fletcher, 1949), though radiologically they appear to be so. The scene is further complicated by Hamlin (1947), who suggests in effect that owing to simple siderosis certain foundry dressers may present radiological findings simulating active silico-tuberculosis, whereas actually they can hardly be classed as even simple silicotics (Riddell, 1948). Sander considers that "the effect of silicosis is primarily on pre-existing tuberculous foci", and that patients with clinically inactive silico-tuberculous lesions can be kept working safely, provided they are examined regularly "at least every six months, including sputum and gastric cultures for tubercle bacilli". In practice —theoretical considerations aside—the main thing is that a silicotic who is allowed to continue in work should be observed periodically by radiological and clinical examination and thorough bacteriological investigation. Sander (1949) and Carozzi (1946) consider that periodical blood sedimentation estimations are most useful in determining whether or not a patient's lesion is becoming "clinically active".

If a silicotic is deemed to have the physical capacity to continue at his present job involving exposure to dust, should he be allowed to do so? Consensus of opinion and evidence would now seem to indicate that in the man aged more than about forty-five to fifty years the disease will follow its appointed course no matter what work he does, and that he will be happier and probably earn more money if he is left in the job at which he is trained. In the case of the younger worker, however, there is some evidence that change from a dusty atmosphere may possibly arrest the disease (Gardner, 1945-1946; Rauschenbach *et alii*, 1948; Greinacker-Oristofari *et alii*, 1947; Vrooman, 1948). Readers who recoil in horror at the idea of allowing an elderly man to continue in a dusty atmosphere would be well advised to consider what becomes of such a man with some skill but little education who is airily advised to "get out of the dust". Often he has to take a "pick and shovel" job and do much heavier work in all weathers for considerably less pay. The sudden change in environment at that age may also inflict psychological trauma from which his spirit never recovers. It is more logical from an industrial hygienist's viewpoint to endeavour to abate the dust and leave the "injured" man where he is than to place in his stead a fresh workman in the same dusty environment who may in turn become "dusted" (Montesano, 1947).

Some silicotics whose physical capacity is failing need change to lighter work. Often the employer provides this without demur. On the other hand, sometimes for the uneducated man this is hard to obtain. When the latter is the case, cooperation of the medical practitioner and Government employment agency officers is obviously indicated. At all events, as a general principle no skilled tradesman should be lightly advised to give up his trade which has taken years to learn until all other avenues

have been exhausted. For the patient who is a tradesman such a decision can be fraught with dire consequences. Unless tuberculosis infection intervenes, pneumonokoniosis is usually more an economic and social than a medical problem. Its main effect is often to reduce the sufferer's capacity for hard manual work, and as a consequence if he is a "pick and shovel" worker he cannot carry on his usual occupation though he is still capable of years of useful work of a lighter nature. The modern trend is to find such a patient a job which he can do rather than to commit him immediately to the dull, slow hours of the "compensation life". This is not always easy, but success is achieved in many cases.

There is also evidence accumulating that fewer than formerly now contract tuberculosis (Sander, 1949; Smith, 1945), and that the number of those who do so bears some rough proportion to the amount of tuberculosis in the community—especially the working community in which the silicotic lives. As medical supervision has pervaded the dusty trades, sources of infection are found and segregated. This is a very cogent argument for regular radiological examinations in the dusty trades.

Remedies.

This article is not concerned primarily with prevention, but a few words on the subject will not come amiss.

With modern methods of dust control available there is really no reason why the foundry industry should continue to produce silicotics. No brief is entertained for those who tend to create a huge dust phobia out of what is a relatively mild hazard; on the other hand, even less time is held for those who either deny that the hazard exists or countenance it because it is relatively mild. In the foundry industry local exhaust ventilation, if properly designed, is efficient and not particularly expensive, and with the exception perhaps of chipping slag in convertors and cupolas the hazardous processes can be made safe. Recent legislation in Britain, in various North American States and in New South Wales confirms this opinion.

SUMMARY.

1. Seventy-six known foundries in Queensland had a "working" population of 1118. Three hundred and eighty-eight of these were considered to have worked sufficiently long in foundries to make the developing of pneumonokoniosis a possibility. Of the 388 foundry workers eligible for the survey, 359 agreed to cooperate.

2. From these 359 patients, occupational and medical histories were obtained; the patients were clinically examined and large chest films were taken. At the same time all foundries were inspected and dust surveys were carried out in approximately 15 of them.

3. Thirteen cases of silicosis were discovered, and in addition eight were eventually considered "doubtful". Ten of the silicotics were considered to have "early" lesions.

4. In the great majority of "positive" cases the subjects were elderly, were foundry dressers or furnacemen and came from the larger foundries. As far as Queensland is concerned, the findings suggest that "dressers"—particularly steel "dressers"—and furnacemen, particularly in large foundries, are exposed to the worst dust hazards.

5. Results of other surveys are discussed, and from this it would appear that the Queensland incidence is not high.

6. Difficulties in the diagnosis of early cases are mentioned, and a plea is made in such circumstances to estimate carefully the industrial exposure which has been experienced.

7. It is contended that it is wrong to regard the man with early silicosis as an invalid.

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SOME PRACTICAL ASPECTS OF VENEREAL DISEASE.

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Hobart.

WHEN I was in Japan as a medical officer with the British Commonwealth Occupation Force, a large proportion of my time was employed in treating venereal disease. Some of the results of this and subsequent experience may be useful to the general practitioner, and certain aspects of syphilis and urethritis are discussed from this point of view.

SYPHILIS.

Diagnosis.

Syphilis may be diagnosed by dark-field examinations, serological tests and clinical observations.

Serological Tests.

Effect of Time.—In my experience, after infection it takes at least three weeks, and not more than three months, for the results of tests to become positive, provided the patient has not received penicillin.

Effect of Penicillin.—I think that penicillin in doses up to 500,000 units lengthens the incubation period of syphilis, but not usually beyond six months. However, I do not doubt that cases could occur in which the incubation period under these circumstances is longer than six months, and the disease may be arrested for one or two years. A large initial dose, say 2,000,000 units or more, not infrequently used in the treatment of urethritis, may kill an associated syphilitic infection; nevertheless, the masking effect of penicillin on the development of sero-positive syphilis is not fully known. On the other hand, although penicillin will delay the appearance of positive results in serum tests, I believe that in cases of established syphilis, in which the results of serum tests have been persistently negative, it can provoke positive results. The following case appears to illustrate this action:

A patient had for several days a rash, which appeared to be a syphilitic one. There were enlarged glands in his neck, axillæ and inguinal regions, and apparently a primary lesion of his finger. The results of Wassermann, Kline and Kahn tests were negative. Attempts to isolate *Treponema pallidum* from the lesion of his finger, and from macules on his arm, were unsuccessful. The serological tests were repeated frequently for four weeks, but the results remained negative, nor were they altered by injections of neoarsphenamine. There was no laboratory confirmation that the patient had syphilis, but his health was deteriorating, and penicillin treatment was begun.

This caused the rash to become temporarily more vivid. The patient's temperature was unaffected (probably because the previous injections of neoarsphenamine had damaged the spirochætes sufficiently to prevent a Herxheimer reaction), and the results of serum tests, carried out on the third day after exhibition of penicillin, were positive and remained so while the patient was in hospital.

It is my impression from this and other cases, that penicillin, in doses of 200,000 or 300,000 units, more effectively provokes positive results from serum tests in patients who have sero-negative syphilis (other than primary syphilis) than does arsenic in the doses recommended for the purpose.

Negative Results from Serum Tests.—The case cited is an example of secondary syphilis with negative serological findings; rare as these cases are, they do occur. Other manifestations of syphilis, for example anal fissures, may exist for many weeks without positive serological findings, and later forms of syphilis may be associated with negative serological findings. Once a case is suspected to be syphilitic, one must not be satisfied with one, two or sometimes more negative results from serum tests, and where these have been constantly negative, I would recommend a provocative dose of penicillin (200,000 units). When one is consulted by an anxious patient because he recently ran the risk of infection, and examination shows no sign of syphilis, negative results from serum tests of one blood sample are not sufficient to exclude syphilis. It is wise after the first blood examination to have further tests performed at monthly intervals for three months, and final tests six months after the time of exposure. In cases in which positive results are found in a sample of blood, a further sample should be taken and serological tests repeated.

Biological Falsely Positive Reactions.—Falsely positive results from serum tests may occur in people who do not have syphilis. I have found the Wassermann test more liable to this error than the Kahn or Kline test, probably because of its more complicated technical procedure, and because of the occasional use of over-sensitive antigens. After antiencephalitis inoculations in 1947, I saw many people with positive results from Wassermann tests, but negative results from Kahn and Kline tests. These findings from Wassermann tests remained positive for some weeks and caused much anxiety, but when a fresh antigen from Australia was used, the results were negative. If the result of a serum test is suspected to be falsely positive, further serum tests should be performed one and two weeks later, and if the results of these tests are doubtful, thereafter at least once a month for six months, after which it should be possible to make a diagnosis. Several conditions besides anti-encephalitis inoculations can cause falsely positive reactions: acute infections, malaria, vaccination for smallpox, tetanus toxoid used as a "booster", infectious mononucleosis, atypical pneumonia, and other diseases, such as leprosy. Apparently, pregnancy does not cause falsely positive reactions. Falsely positive reactions are usually present for only three to twelve weeks, and generally are weak. Except in pregnancy, a person with a positive serum reaction only does not require immediate treatment. In pregnancy, treatment should be given without delay. One should remember that of all patients with latent syphilis, no more than 25% if untreated develop disabling or fatal disease, and that latent syphilis is not contagious. In those cases in which doubt exists, examination of the cerebro-spinal fluid should be performed.

Imperceptible Invasion.

I recall one patient who, after a single exposure, was frequently examined and assured that he had not contracted syphilis. After nearly a year's reassurance, someone had serum tests carried out, and the results were positive. This patient did not have a primary chancre, or apparently any other clinical sign of syphilis during the invasive period. The disease can establish itself without any notice of its attack.

Treatment.

Jarisch-Herxheimer Reaction.

When the *Treponema pallidum* is destroyed in the tissues by antisyphilitic treatment, foreign proteins are liberated, the organisms may temporarily become more active, and increased inflammation results in the infected tissues. This aggravation shows itself as a clinical reaction called the Jarisch-Herxheimer reaction.

The reaction usually subsides in twelve hours. A simple general reaction occurs in almost every syphilitic who is treated with penicillin, and for whom no treatment to prevent the reaction has been given. The patient complains of headaches, pains behind his eyes and malaise. His temperature rises, and he may have rigors. All these symptoms and signs develop six to eight hours after the initial injection. This syndrome, when the reaction is at all severe, so closely resembles malaria that in the first few cases I encountered I had blood films examined for malarial parasites; none were found.

However, the reaction may show itself only locally. Thus when only one lesion is obvious, such as a primary chancre, the result on the giving of penicillin is pain, swelling and redness in the sore. But even in these cases, if the infection is well established, the general systemic reaction may considerably upset the patient. If vital structures should be severely infected, for example, the basilar artery or the orifice of the coronary artery, increased inflammation in these tissues could cause a fatality.

There is, I think, a third form of reaction, for which the name erythrogenic Herxheimer reaction seems appropriate. It occurs in cases of secondary syphilis in which the rash has not yet appeared, but positive serum reactions are obtained.

I first saw it in a patient undergoing treatment with penicillin given at three-hourly intervals for gonorrhœa. I was called to see him after his third injection, because he had developed a rash, which the nursing orderly thought was a penicillin reaction. The rash was red, evenly distributed in macules a quarter to half an inch wide over the whole body, and not itchy. It resembled a typical secondary syphilitic rash. The patient had the symptoms and signs of a simple Herxheimer reaction, that is, clinically his condition resembled malaria. Two days later the results of serum tests, carried out on a sample of his blood taken just before the penicillin was given, were available. They were positive. I think that penicillin had caused the spirochætes in the throes of death to produce their erythrogenic toxin, causing a secondary syphilitic rash to appear before it would have done so in the ordinary course of the disease.

In my experience, general Herxheimer reactions (and I regarded patients as having one only if their temperature was 100° F. or more) occurred in 25% of patients with primary syphilis, in 55% of those with secondary syphilis (due to more severe infection) and, because of several factors, in a smaller percentage of those with latent syphilis. A Herxheimer reaction, occurring in a patient with unsuspected latent syphilis being treated for, say, pneumonia, could cause some worry if its true nature was unrecognized. It can be diagnosed only if one knows it exists.

Steps which should be taken to avoid this reaction will be described. Treatment, beyond "Nembutal" three grains and "A.P.C." mixture one and a half ounces given together as often as needed to alleviate the patient's discomfort, is not usually needed. If a patient had a severe reaction, being in some distress, with a temperature of 103° F. or more, injections were suspended for twelve hours, but I did this only when the reaction was severe. When injections were stopped, the patient's temperature came down, and his headaches and rigors ceased. Reinstitution of penicillin therapy in full doses after twelve hours was always uneventful.

It seemed to me more humane, with a severe reaction, to do this, but several writers state that the correct procedure is to continue with penicillin administration and the reaction will settle down. Certainly less severe reactions, in which the temperature did not exceed 103° F., settled down while penicillin injections were continued. When penicillin in oil is being used, this view is a comfort. If precautions are taken in cases in which one might expect a serious reaction, for example, in treatment of cardio-vascular syphilis, it should not cause any deaths.

Treatment with Penicillin, Arsenic and Bismuth.

In Primary and Secondary Syphilis.—Preferably the patient with primary or secondary syphilis should be given crystalline penicillin 50,000 units three-hourly to a total of 4,000,000 units. Penicillin K seems to be less effective than penicillin X or G. On the second day of treatment 0·2 gramme of bismuth subsalicylate should be given by intramuscular injection and 0·3 gramme of neoarsphenamine or 0·03 gramme of "Mapharsen" by the intravenous route. On the fifth day 0·45 gramme of neoarsphenamine or 0·045 gramme of "Mapharsen" should be given. On the eighth day 0·2 gramme of bismuth subsalicylate and 0·6 gramme of neoarsphenamine or 0·06 gramme of "Mapharsen" are given, and these doses are repeated at weekly intervals for a further eight injections. But this method of penicillin administration requires the patient to be in hospital. For ambulatory patients I have used 300,000 units of penicillin in oil daily, combined with an injection of 100,000 units of watery solution twice daily for eight days, and when the patient will stand it, for ten days. Arsenic and bismuth were used as described in the previous paragraph. This method seems satisfactory, but I have not treated a sufficient number of patients for a long enough time to speak categorically.

In Latent Syphilis, Neurosyphilis and Cardio-Vascular Syphilis.—Patients with latent syphilis and with neurosyphilis should be given 0·1 gramme of bismuth and three days afterwards a further 0·2 gramme. Three days later 0·3 gramme of neoarsphenamine should be given, and if within the next three days no reaction occurs, penicillin up to at least 6,000,000 units, combined with arsenic and bismuth treatment as previously described, should be given. However, if a reaction is still feared after administration of neoarsphenamine 0·3 gramme, injections of arsenic and bismuth in the doses prescribed for treating primary and secondary syphilis should be given before carrying on with penicillin treatment. When neoarsphenamine 0·6 gramme and bismuth 0·2 gramme can be given without suggestion of a Herxheimer reaction, penicillin may be exhibited. Most patients with early neurosyphilis, such as syphilitic meningitis, treated with penicillin, arsenic and bismuth, should respond satisfactorily. But if this form of treatment is not effective within six months, or if the condition is one of advanced neurosyphilis, pyrexial therapy, in addition to antisyphilitic drugs, should be given. Cardio-vascular syphilis should be treated with potassium iodide, grains five, gradually increased to grains fifteen three times daily, combined with weekly injections of bismuth (0·5 gramme initially gradually increased to 0·2 gramme) for twelve weeks. One then proceeds as for latent syphilis. If clinically a too vigorous attack is suggested, one should proceed more slowly with arsenic injections before exhibiting penicillin, initially with small doses.

Surveillance.

Surveillance is vital to successful treatment, as probably relapse will occur in 5% or more of cases, depending on whether the condition is early or late syphilis. Wassermann tests and Kahn (quantitative) tests should be performed two, four, six, nine, twelve, eighteen and twenty-four months after completion of penicillin treatment. A definite and continued change in the titre of the Kahn tests will be seen if the infection is subsiding or relapsing. Six and eighteen months after penicillin treatment, clinical examination of the patient, together with full examination of the cerebro-spinal fluid, should be carried out. I think that this is a minimum period of surveillance; and serum tests should be performed yearly for a further three years. Recrudescence after this period is unlikely.

Patients who have been recently treated for syphilis, or who are still under treatment at out-patient depart-

ments, should not be given penicillin for other conditions, unless it is absolutely necessary. If additional penicillin is given, surveillance should be continued from the time of this dosage for two years, as it was previously; but if cerebro-spinal fluid examinations have been carried out, I feel that it is unnecessary to repeat them unless involvement of neural tissue is suspected. Most patients who are under treatment for syphilis, or who have had it recently, do not volunteer the information to the doctor treating them for some other disease. If any fact of his past life suggests that a patient may have contracted this disease, it is worth while questioning him.

GONOCOCCAL AND NON-GONOCOCCAL URETHRITIS IN THE MALE.

Diagnosis.

Clinically, gonococcal and non-gonococcal urethritis are indistinguishable, although gonococcal urethral discharges are often more profuse, and respond better to treatment, than do non-gonococcal ones.

Cause.

Many different things can cause urethritis. Excluding those cases occurring after prophylactic irrigations, frequent self-examination and the introduction into the urethra of substances like toothpaste, I have never encountered a case of urethritis (including cases of abacterial pyuria and, I think, one of Reiter's syndrome) that I was quite certain had not resulted from sexual intercourse.

Gonococci have caused about half the number of cases of urethritis I have encountered; the remainder, together with those produced by the agents mentioned, have apparently been caused by staphylococci, streptococci, Gram-negative cocco-bacilli, *Bacterium coli*, diphtheroids, and probably other organisms I have not recognized. In many cases, no organisms were seen.

Treatment of Uncomplicated Urethritis.

The routine treatment of gonococcal and of non-gonococcal urethritis is the same. At the first visit, a smear of the discharge is taken for subsequent microscopic examination, and the patient is given penicillin and one of the sulphonamides. I would recommend procaine penicillin 300,000 units together with 100,000 units of crystalline penicillin, the latter being repeated twelve hours later. If the second injection of 100,000 units of penicillin is impracticable, then 200,000 units of penicillin should be injected at the same time as the oily solution.

A sulphonamide in routine doses to a total of 25 grammes should be given. In my experience it does not seem to matter what sulphonamide, or mixture of sulphonamides, is prescribed, although I have felt that sulphadiazine is the most effective. An alkaline mixture should be prescribed, and the patient instructed to drink about eight pints of fluid daily, but to avoid alcohol for at least a month.

It is most important to impress upon the patient not to examine his meatus and run his finger along his urethra to see if he still has a discharge. This procedure prolongs his urethritis; if he has an appreciable discharge it will be quite obvious to him without performing any manipulations. Anxiety is usually a predominant symptom, and one should tell him at the first visit that with treatment the condition will clear up quickly, providing he does as advised. He should be told that, if complications are present, some weeks may pass before complete healing occurs.

At this first visit, blood should be taken for serological examination. Occasionally one finds that the patient has contracted syphilis in the past; naturally serum tests do not show the presence of a recent infection, unless it is older than twenty-one days. If there is an ulcer on the penis, then penicillin should not be used; a sulphonamide should be given alone, otherwise a concomitant syphilitic infection may be hidden.

The patient should be told of the possibility that he may have contracted syphilis with his urethritis, and that it is necessary to take blood from him for serum tests once a month for three months, thereafter every three months for twelve months, to exclude this possibility.

The day after the penicillin injections the patient should be examined again, and if the condition appears to have subsided no further penicillin is given, although he should be told to return if there is an obvious recurrence. About fourteen days after the penicillin treatment, the prostate should be examined digitally, and a prostatic smear taken. The patient should pass a specimen of urine, and this should be viewed macroscopically for debris or prostatic threads. A curved sound should be passed for evidence of stricture or littritis. If, on the following morning, before passing his urine, the patient notices a discharge, he should be instructed to dab it with a glass slide for subsequent examination. These examinations (full test of cure) should display any abnormality, which can then be treated. Full test of cure should be repeated three months after the disease seems to have been cured. A convenient time is when blood is taken for the third monthly serological examination.

But if after one day's treatment the condition has not subsided, a second dose of penicillin of the same size should be given, and so on each day. If treatment occupies some days, urethral smears should be taken, and an early morning specimen of urine examined macroscopically every alternate day. Penicillin administration may be continued up to a total of 4,000,000 units; in my experience, if the condition does not clear up fairly well in seven days, continuation of penicillin does not greatly assist progress. On the fourth day of treatment in a resistant case, I think that very light prostatic massage (as one would make an examination *per rectum*) should be given, and thereafter light prostatic massage should be given about once every three days. If, in a further seven to ten days, there is little improvement, then with a 20-millilitre syringe, gentle irrigations with a warm solution of potassium permanganate 1:8000, or other solutions such as silver nitrate 1:10,000, mercuric oxycyanide 1:10,000, or normal saline, may be given daily for three days, then on alternate days. Some patients will not tolerate one solution, but will another. Zinc sulphate 1:1000, an astringent, will sometimes clear up a small persistent discharge. If the condition appears to have become stationary, a medium-sized sound can be passed, and this procedure may be repeated at intervals of four days. Light prostatic massage should be continued. However, one must be careful not to overtreat; too many irrigations or interference with sounds will maintain urethritis. The condition will improve, very gradually, if the patient drinks plenty of fluid and has no other treatment.

Usually these resistant cases occur when penicillin has been withheld because of a strong suspicion of syphilis. If, after reasonable trial with sulphonamides and the procedures mentioned, say for two weeks, no response occurs, penicillin is given and surveillance instituted as for syphilis, but excluding cerebro-spinal fluid examinations unless suggested clinically.

Complications of Urethritis.

Diagnosis.

With the exception of prostatitis and littritis, complications are easy to recognize. Palpation of the urethra over a sound will disclose the small lumps indicating littritis, but the recognition of prostatitis is more difficult. This is diagnosed when examination through an oil-immersion lens of an evenly distributed prostatic smear shows more than nil to four pus cells per field. The smear is taken after the patient has passed urine, and if doubt exists after examination of a smear, further smears are taken at intervals of one or two days until a decision is made. Organisms are seldom seen in a prostatic smear. For ordinary purposes staining with methylene blue is quite satisfactory. Digital examination of the prostate *per rectum* is necessary, but little reliance should be placed on the findings. The examination of smears is essential. However, digital examination helps to judge the progress of treatment.

Treatment of Prostatitis and Other Complications.

Prostatitis.—Experience in treating prostatitis with penicillin alone, then with penicillin and prostatic massage, convinced me that prostatic massage must be done to clear up a well-established infection. It promotes drainage and a flow of fresh blood to the gland, but I avoid its use as much as possible, as epididymo-orchitis not infrequently appears during a course of prostatic massage. Patients with mild prostatitis, when examination of smears shows only perhaps one to eight pus cells per oil-immersion field, can be left untreated for about six weeks; then further smears are examined, and if the condition has not resolved, treatment is given. When a well-established infection exists, penicillin 2,500,000 units should be given and prostatic massage employed twice weekly for two weeks, thereafter once weekly for at least a month, when a smear should be examined. After each massage the patient should be given two glasses of water to drink. In refractory cases, twice-weekly massage may be necessary for some time. Usually treatment takes two to six months to clear up a well-established infection completely. The treatment of prostatitis is important, since although the patient is usually without symptoms or signs, an infection will persist for many years, and may eventually cause joint or genito-urinary disease.

Other Complications.—Littritis is treated by massage of the palpable glands, once every two or three days over a sound, and continued until the inflammation subsides. If this is not successful in a month, other procedures requiring the use of a urethroscope may be necessary. Successful treatment is shown by inability to palpate the glands, and absence of symptoms or signs. This condition seldom causes much trouble. If the patient develops epididymo-orchitis, he should be confined to bed for at least a week with his testicle supported by a suspensory bandage, and as a routine 2,500,000 units of penicillin are given. The suspensory bandage should be worn until all signs of inflammation have subsided, which usually takes about three months. Prostatitis is often present, and should be treated. Other complications are treated as one would treat similar conditions arising from other causes.

SUMMARY.

1. Negative laboratory findings in a case of suspected syphilis must be accepted with reserve.

2. A general Herxheimer reaction resembles an attack of benign tertian malaria, occurs within twelve hours of initiation of treatment with penicillin in many cases of syphilis, and usually subsides in twelve hours.

3. An ambulatory method of treating syphilis with penicillin, bismuth and arsenic is described.

4. Identification of the organism causing urethritis is not important in its treatment.

5. Prostatitis commonly complicates urethritis, and often remains after the urethritis has subsided. It is diagnosed by examination of a prostatic smear, and prostatic massage is essential in treatment.

6. An ambulatory method of treating urethritis is described.

7. Blood serological tests and cerebro-spinal fluid examinations should be carried out for two years after penicillin treatment of syphilis; and serological tests should be carried out for one year after penicillin treatment of urethritis, in order that relapsing or developing syphilis may be discovered.

ACKNOWLEDGEMENT.

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Morgan, Hugh J. (19—), "Syphilitic Aortitis and Aneurysm", in Cecil's "Text Book of Medicine", Seventh Edition, page 396.

Reviews.

A YEAR BOOK OF NEUROLOGY, PSYCHIATRY AND NEUROSURGERY.

THE section on neurology in "The 1949 Year Book of Neurology, Psychiatry and Neurosurgery" has a new editor, Roland P. Mackay, Professor of Neurology in the University of Illinois.[1] His credo, expressed in the introduction, is "that all science is one, that its multifarious fields are divided only by differences in their subject matter, their methods of approach and their variety in emphasis". Similarly, he states that "all medicine is one, and neurology, the study of the nervous system in health and disorder, can disregard no portion of the rest of the medical field". He is well aware of the unhappy cleavage that can appear between neurology and psychiatry, to the loss of both, and seems determined to discourage it. All this suggests that he will be a sound member of the editorial team of this Year Book. The material in the section on neurology, which the editor clearly designates as representative and not exhaustive, is grouped into chapters on anatomy and physiology, pathology, trauma, infectious diseases (meningitis, encephalitis and myelitis, syphilis), degenerative diseases, vascular encephalopathies, convulsive disorders, the neuropathies, exogenous toxins, and therapeutic agents and methods.

Nolan D. C. Lewis continues as editor of the section on psychiatry. In his introduction he expresses views largely complementary to those of Mackay, and like him refers to the stimulating effect upon medical thought of the psychosomatic approach. The abstracts in this section are grouped into chapters on general topics, child psychiatry, schizophrenia, affective disorders and miscellaneous reactions, organic disorders and toxic reactions, psychoneurosis and psychosomatic disorders, and therapy.

The section on neurosurgery is again edited by Percival Bailey, who uses his introduction to give a brief but informative survey of the changes and advances throughout his field. This practice of making a survey of the subject is adopted by only a limited number of Year Book editors; many readers would probably welcome its adoption by others. The introduction to the section on neurosurgery would serve as a model. The chapters in the section deal with brain tumours, trauma, sympathetic nerves, motor disorders, peripheral nerves, herniated disks, lobotomy, epilepsy, electroencephalography, angiography, vascular anomalies, malformations, infections, technique, air injections, pain and miscellaneous subjects.

This is a large volume and many journals have been scanned to compile it; they were those received by the editors between December, 1948, and October, 1949. Eleven contributions included are from Australian authors, and Continental literature is well represented, as well as that of the United Kingdom and North America. Most practitioners will find something of interest and value in this Year Book. As Lewis states in the introduction to the section on psychiatry, "we are in a 'psychiatric era' in medicine with all its desirable and undesirable features and results; the medical specialist as well as the general practitioner will do well to keep himself informed on the major issues".

A YEAR BOOK OF ORTHOPÆDICS AND TRAUMATIC SURGERY.

THE principle adopted in the compilation of "The 1949 Year Book of Orthopedics and Traumatic Surgery", as stated by its editor, Edward L. Compere, was to attempt to select for abstracting every article which in the editor's opinion would be of interest and help to medical practitioners who wish to keep abreast of the significant literature reporting advances or reaffirming approved principles in orthopædics and traumatic surgery.[2] The journals reviewed for this purpose were those received by the editor between October,

[1] "The 1949 Year Book of Neurology, Psychiatry and Neurosurgery (December, 1948-October, 1949)"; Neurology—edited by Roland P. Mackay, M.D.; Psychiatry—edited by Nolan D. C. Lewis, M.D.; Neurosurgery—edited by Percival Bailey, M.D.; 1950. Chicago: The Year Book Publishers, Incorporated. 7" × 5", pp. 668, with 113 illustrations. Price: $5.00.

[2] "The 1949 Year Book of Orthopedics and Traumatic Surgery (October, 1948-November, 1949)", edited by Edward L. Compere, M.D., F.A.C.S.; 1950. Chicago: The Year Book Publishers, Incorporated. 7" × 5", pp. 464, with 238 illustrations. Price: $5.00.

1948, and November, 1949. An interesting selection has resulted. The number of papers on plastic surgery of the trunk and extremities, plentiful in the war and immediate post-war period, is now so small that the chapter on the subject has been discontinued. Papers on bone and joint tuberculosis were also few and have been included in the section on osteomyelitis and other infections. Orthopædic conditions related to metabolic disturbances and malnutrition are grouped in the chapter entitled "Miscellaneous". A new chapter deals with the embryology, physiology and anatomy of the skeletal system; it contains material of considerable interest and adds to the value and standard of the Year Book. Bone banks are considered so important as to warrant mention in the title of the chapter in which they are included with instruments and appliances. The other chapters are much as before. That on poliomyelitis is surprisingly short, containing only three abstracts. Congenital deformities are given good space, the chapter being marked by several interesting European contributions. A short chapter on the epiphyses is followed by one on osteomyelitis and other infections that should be of general interest. The chapter on tumours, cysts and fibrodysplasia contains a number of valuable contributions including a discussion of osteogenic sarcoma as a complication of *osteitis deformans*, by H. R. Sear, of Sydney. The length of the chapter on arthritis and rheumatism reflects the growing interest and advances made in the treatment of these long-neglected conditions. Fractures, dislocations and sprains naturally have a prominent chapter, which contains some new material. Other chapters are devoted to amputations and prostheses and to surgical technique, and the remaining abstracts are grouped anatomically. The orthopædic surgeon will find much to interest him in this volume, as will the general surgeon and the general practitioner with a taste for orthopædics. Certain sections, for example that on rheumatism and arthritis, will attract a wide circle.

POPULAR ANÆSTHETICS.

EVERYTHING considered, Gordon Ostlere's little book "Anæsthetics and the Patient" is a commendable effort.[1] The author has faced no easy task in making his presentation suitable for lay persons, and yet avoiding the creation of false impressions in the minds of nurses and junior medical students, for whom it is also recommended.

The work opens with a somewhat lurid account of the sufferings endured by the victims of surgery before the introduction of general anæsthesia. A brief historical survey follows, in which due credit is accorded to Morton as the essential discoverer of ether anæsthesia. The initial trial of nitrous oxide by Horace Wells, however, is recorded in terms which differ from those of the accepted story. Perhaps the journalistic style employed by the writer demands such licence.

Anæsthetics are classified under the three headings of general, local and spinal, following which their modes and sites of action are dealt with in some detail. Useful, if simple, diagrams accompany this elementary yet informative discussion. Next the recognized stages of anæsthesia are described with the help of various homely comparisons.

The several anæsthetic agents, premedication and apparatus then receive brief attention. Due notice of the trials and efforts of such pioneers as Colton, Simpson, Snow and Clover occurs in these sections. Here a remarkable error is perpetrated; John Snow, born in 1813, is said to have been a pupil of John Hunter, who died in 1793! The truth is that Snow attended the Hunterian School of Medicine in Windmill Street, London, from which he graduated in either 1836 or 1838.

Modern apparatus and methods, including further details about local and spinal analgesia, next come under review. A good chapter on the scope and limitations of anæsthesia in midwifery precedes a brief assessment of curare. Here there is a strange omission; the fact that H. King, of London, was the first to isolate the active principle of curare, *d*-tubocurarine chloride, is not mentioned.

With some note of the main complications of anæsthesia, a review of modern procedures especially in relation to lung and brain surgery, and a few reassuring admonitions to the reader, the book closes with a brief but adequate index. Its large and clear type, the absence of printer's errors, and its lively style all make for easy reading and sustained interest.

[1] "Anæsthetics and the Patient", by Gordon Ostlere, M.A., M.B., B.Chir., D.A.; 1949. London: Sigma Books, Limited. 7¼" × 4⅝", pp. 170, with a few illustrations. Price: 7s. 6d.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Atlas of Human Anatomy: Descriptive and Regional", by M. W. Woerdeman, M.D., F.R.N.A.Sc.; Volume I; 1948. London and Australia: Butterworth and Company (Publishers), Limited. 9½" × 7", pp. 500, with 512 illustrations, some of them coloured. Price: 82s.

This volume deals with osteology, arthrology and myology.

"Postgraduate Obstetrics and Gynæcology, 1950", by F. J. Browne, M.D. (Aberdeen), D.Sc., F.R.C.S. (Edinburgh), F.R.C.O.G.; 1950. London and Australia: Butterworth and Company (Publishers), Limited. 9½" × 6½", pp. 552, with 107 illustrations. Price: 65s.

Selections from talks given by the author to doctors specializing in obstetrics and gynæcology.

"Osteology for Dissectors: A Tutorial Pocketbook", by Robert King Howat, M.B., C.M. (Glasgow), F.R.C.S. (England), F.R.F.P. and S. (Glasgow); 1950. London: Henry Kimpton. 7" × 5", pp. 292, with 46 illustrations. Price: 15s.

Designed for use with the dry bones of the parts under dissection.

"Facial Paralysis: Being a Treatise on a Clinical Classification of Paralysis of the Facial Nerve", by J. Parkes Findlay, M.B., Ch.M.; 1950. Published by the author and distributed by Angus and Robertson, Limited. 9½" × 6", pp. 48, with illustrations.

The full account of work published in shortened form in this journal on February 11, 1950.

"Human Embryology for Medical Students", by Dr. S. R. Nair; 1950. Bombay: The Popular Book Depot. 8½" × 5½", pp. 416, with 241 illustrations. Price: £2 2s.

An attempt to produce a history of the development of the human embryo, with no reference to comparative anatomy.

"Penicillin: Its Practical Application", edited by Professor Sir Alexander Fleming, M.B., B.S., F.R.C.P., F.R.C.S., F.R.S.; Second Edition; 1950. London and Australia: Butterworth and Company (Publishers), Limited. 8½" × 5½", pp. 506, with 63 illustrations. Price: 42s.

The second edition of a work first published in 1946.

"A Story of Nutritional Research: The Effect of Some Dieting Factors on Bones and the Nervous System", by Sir Edward Mellanby, G.B.E., M.D., Sc.D., F.R.S.; 1950. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 8" × 5¼", pp. 462, with 73 illustrations. Price: 53s. 9d.

The Abraham Flexner Lectures which were to have been given in 1941.

"Practical Neurological Diagnosis: With Special Reference to the Problems of Neurosurgery", by R. Glen Spurling, M.D.; 1950. Illinois: Charles C. Thomas (Publisher). Oxford: Blackwell Scientific Publications, Limited. 9" × 6", pp. 288, with 101 illustrations. Price: 37s. 6d.

Designed "to present a simple account of the principles of neurological diagnosis" for students and practitioners.

"Principles and Practice of Plastic Surgery", by Arthur Joseph Barsky, M.D., D.D.S.; 1950. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 10" × 7", pp. 512, with 1029 illustrations, some of them coloured. Price: £5 7s. 6d.

"This book is intended to be a practical guide, not an arm chair surgery, and no effort has been made to be all-inclusive."

The Medical Journal of Australia

SATURDAY, AUGUST 5, 1950.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

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CHILDREN AND THE CINEMA.

No one today doubts that the cinematograph has some sort of influence on those who have acquired what has so well been called the "cinema habit". The powers-that-be show that they recognize this by the requirement that films shall be classified as suitable either for general exhibition or for showing to an audience of adults. It is not a simple matter to determine whether the influence of "pictures" is good or bad, or if a bad influence is descried to say how far it extends. Some of those who are interested in the psychology of children declare out of hand that the cinematograph has a pernicious influence on the child mind. Such a statement is easy to make; those who make it should be prepared to justify it, and again this is not a simple matter. Of one fact we may be certain, and that is that the cinema has an enormous attraction to great numbers of people. Since this attraction is likely to grow rather than become less, the subject is worthy of discussion.

In this journal on December 13, 1947, attention was drawn to a "social survey" reported in *The Times* on the "cinema habit" in England. It was stated that of the adult population 32% went to the cinema at least once a week and that 13% went more than once a week. Of children of school age 65% went to the cinema once a week or more and only 5% did not go to the cinema at all. It was estimated that the expenditure of the civilian population at the cinema was something over £100,000,000 a year. In the issue of June 1, 1940, an account was given of the views expressed by Mr. Richard Ford in a book entitled "Children in the Cinema", published by George Allen and Unwin. Ford's general conclusion was that for the vast majority of children the cinema was beneficial rather than harmful, provided care was taken in the selection of films. It was pointed out that the morally questionable element in films was ignored by children of school age, and that love-making on the screen bored the child because he did not understand it. The drama that he did understand, however, was real to him and he identified himself with the character that he was watching. Ford insisted that the process of identification should not be mistaken for suggestion. He held that the child was satisfied with the success of right over wrong and did not feel compelled to copy miscreants. We pointed out that there were some who would not agree with Ford's views. At the same time we suggested that the overwhelming negative evidence presented by Ford in his discussion on the responsibility of films for child crimes supported his contention that the cinema should be absolved from blame.

In a report recently issued in Great Britain by a departmental committee[1] this subject is discussed in an interesting fashion. The committee consisted of 21 persons and Professor K. C. Wheare was chairman. It was appointed to consider and report upon:

(*a*) the effects of attendance at the cinema on children under the age of 16, with special reference to attendance at children's cinema clubs;

(*b*) whether, in the light of these effects, any modification is desirable in the existing system of film classification, the existing position with regard to the admission of children to cinemas, or in the organization, conduct and management of children's cinema clubs.

We do not propose to traverse the whole of this report, but to consider shortly the committee's views on the effects of the cinema, on the cinema and juvenile delinquency and on some of the measures suggested for future action.

No well-established conclusions on the physical effects on children of frequent cinema attendance were advanced, and even in regard to eyesight no positive evidence of harmful effects was forthcoming from experts. Such matters as ventilation, over-crowding, disinfection, cleanliness and so on are mentioned, together with the need for some precautions in regard to them. The nervous effects are stated as likely to be considerable in their immediate manifestations and in some cases to be deplorable. The committee, however, finds itself unable to make a valid statement about the sources of fear in films because children vary so much in their reactions to fright and fear and in the objects which cause these emotions. It thinks that if the matter was scientifically investigated some surprising results, both positive and negative, would appear. At the same time it expresses the opinion that much more care and knowledge than are employed at present should go to the shaping of cinema programmes for children and to the regulation by parents of the frequency of their children's attendance. On the question of the moral effects of films on children the committee appears to be somewhat ill at ease. An attempt was made to obtain specific information, as well as opinions, on the subject. Inquiries were made about the connexion between the viewing of an incident in a film and the appearance of a child before a juvenile court. In a six months' period, during which approximately 38,000 children under the age of sixteen years were brought before juvenile courts, there were only 141 cases in which delinquency, and only 112 in which moral laxity, had appeared to have a direct connexion with an incident in a film seen by the child. In view of these figures the committee thinks that it cannot be said to be proved that cinema attendance is likely to lead to delinquency and moral laxity in children. Though in "a very few cases" certain parallels were

[1] Report of the Departmental Committee on Children and the Cinema; Presented to Parliament by the Secretary of State for the Home Department, the Secretary of State for Scotland and the Minister of Education by command of His Majesty, May, 1950. London: His Majesty's Stationery Office. 9½" × 6", pp 114. Price: 3s. net.

suggested between the themes of crime and vice shown on the screen and the patterns of offences leading to the juvenile court, the link of cause and effect was in most cases unproved and the committee thinks that "subtler and more various influences" are at work. The committee shows its uneasiness by stating that its members are not happy about the moral values portrayed in many films. "If films are seldom vicious they are often crude and stupid and likely to exert a contrary influence to that of a good home and a good school." The committee is not unanimous in its views. One of its members, Mrs. Henrietta Bower, has made her signature of the report subject to reservations set out in a special memorandum. Her reservations deal with the power exercised by the cinema upon children and with the connexion between juvenile delinquency and cinema attendance. She makes the following categorical statements: (i) The cinema is a very formidable power for good or evil, and the most potent single means for influencing the great mass of people, including children. It gives equal force and attractiveness to what is good, bad or indifferent. (ii) The cinema has a greater power than any single medium to provoke a child to imitation, and an almost uncanny power of making the unreal appear real. There has been considerable evidence that many children up to nine or ten think that the film is real. (iii) The cinema also induces a predisposition of mind, especially as to what is right and what is wrong, what is fashionable and what is not. A child is thus led to indulge its inherent tastes, for example, for knowledge, for violence, for pleasure, or to be in the fashion. (iv) The cinema promotes mass reactions in children, as observed in an uninhibited form at children's cinema exhibitions. Mrs. Bower holds that the minds of children are elevated or degraded momentarily according to the theme and incidents of the film portrayed. She cannot accept the conclusions of the rest of the committee that the cinema is not a major direct cause of juvenile delinquency. To her the conclusion is inescapable that the cinema is in fact an important contributory cause, particularly where cinema going is excessive. It is to be noted that Mrs. Bower refers to a contributory cause and not a major cause. She makes a good point when she states that if a child goes to a cinema three times a weak, he will during the period of five to fifteen years of age have 1560 attendances. There will thus be a good deal which he will have to absorb, which he will have to resist, or to which he will have to adjust his mind.

This whole discussion is necessarily somewhat academic and conclusions reached must be largely based on the opinions of individuals who may prejudge the problem before they tackle it. We all assent to the use of films for certain propaganda purposes, such, for example, as the dissemination of information and tuition on matters of health. It would be absurd to accept the cinema for propaganda on health and to deny that it can influence the minds of children in other directions. Any effect that cinematograph films have will depend on the receptivity of the minds of those who see them, on the ideas to which, in the language of physiology and psychiatry, they are conditioned, and on the type of film. If the cinema is to be an influence for good, or if, at a lower level, it is not to do harm, we, as a community, must not only try to bring about a proper, a strict, classification of films in the interests of children and regulate their attendance, but also strive for a proper conditioning of the minds of children, a matter of home life and education. The committee issuing the present report has published an appendix setting out suggestions for research in this matter and this is to be recommended, for after all reliable knowledge is the only basis for action. The main problem is the creation in the community of a proper sense of values. This is an enormous subject and to many no doubt hopeless. But it would be foolish to be defeatist in the matter. A sphere of action is waiting in schools and other institutions. The number of people to whom medical practitioners can talk on this, and indeed on any, subject is considerable and they can influence home life more than any other group of persons. In any case the more the problem is discussed, the better; in this way public opinion is formed.

Current Comment.

PROPHYLACTIC INOCULATIONS AND POLIOMYELITIS.

THE reports from Victoria earlier this year of a possible relationship between prophylactic inoculations and the incidence and distribution of paralysis in poliomyelitis have caused a good deal of anxiety among the general public and some uneasiness among medical practitioners. The facts of the matter so far apparent, though cause for serious thought, are not so alarming as first impressions may have suggested. The evidence has been presented by B. P. McCloskey,[1] of the Victorian State Health Department, from investigation of 340 cases of poliomyelitis (250 being personally investigated by McCloskey) following suggestions from a small number of parents that the development of poliomyelitis by their children was related to prophylactic inoculation. When the investigation was undertaken, any real association between poliomyelitis and inoculation was considered highly improbable, but the evidence obtained, McCloskey considers, indicates that an injection of pertussis vaccine, given during an epidemic of poliomyelitis, may determine the onset of paralysis in the immunized child; the evidence that an injection of diphtheria toxoid may have similar effects is, he feels, less conclusive. A particularly striking feature apparent from his data is that paralysis was much more frequent in the inoculated than in the uninoculated limb after injection of pertussis vaccine alone or in combination; the phenomenon was again not so striking with diphtheria toxoid. Explanation of the findings is not easily made. McCloskey presents convincing reasons for ruling out contamination of the prophylactic agent with poliomyelitis virus and syringe transmission, apart from the fact that these possibilities are not in harmony with current views on poliomyelitis. He allows that it is conceivable that, in a subject suffering from non-paralytic infection, circulating virus might be arrested and concentrated in tissue damaged by the vaccine, and then travel, perhaps after multiplication, via the peripheral nerves or some other channel, to the corresponding areas of the spinal cord; here, however, it is necessary to postulate the occurrence of viræmia (an idea by no means accepted by virologists), and this at the time of the injection or before the injection damage has been repaired. The last hypothesis considered is the suggestion of D. M. Horstmann and J. R. Paul,[2] in explanation of the effect of exercise and other forms of trauma on the development of paralysis, that local peri-

[1] *The Lancet*, April 8, 1950.

[2] *The Journal of the American Medical Association*, September 6, 1947.

pheral trauma may be reflected in corresponding areas of the spinal cord in the form of central changes which favour activation of virus already present in the central nervous system. This possibility has something to commend it, but McCloskey is not convinced by any of the explanations suggested and puts forward ideas for further experimental investigation.

The matter came in for vigorous discussion, according to the report,[1] at a meeting of the section of epidemiology and State medicine of the Royal Society of Medicine on April 21, 1950. F. M. Burnet, of Melbourne, is reported to have contended that McCloskey had clearly shown that paralytic poliomyelitis in the injected limb occurred within two months of inoculation with pertussis vaccine, either alone or in combination with diphtheria toxoid, more often than would be expected by chance, most cases arising within seven to eleven days of the injection—that is, within the accepted limits of the usual incubation period of poliomyelitis. Burnet considered less conclusive the evidence incriminating diphtheria toxoid given by itself, but there was a suggestion of increased frequency of paralysis in the injected limb five to fourteen days after inoculation with this agent. Subcutaneous or intramuscular injection of these antigens seemed to be followed by a period of heightened vulnerability to the paralytic effects of poliomyelitis in the injured limb, which lasted usually for seven to eleven days but might last up to forty days. The possibility of contamination of needle or prophylactic agent was ruled out as an explanation of the phenomenon by Burnet, and he did not appear to be impressed by the ideas that the injection site provided a suitable milieu for the multiplication of virus circulating in the blood, the virus then moving up a neuronal path to the central nervous system, or that some toxic agent in pertussis vaccine, when deposited in or near a muscle, tracked up the motor nerve in something of the manner of tetanus toxin. He inclined to the belief that the inflammatory reaction produced fibrosis and scarring, which affected the nerve trunks and increased their vulnerability by producing a reflex neural disturbance in the corresponding region of the spinal cord. The effect, he thought, was analogous to that of severe exercise.

In the ensuing discussion, A. Bradford Hill presented the preliminary results of an English survey, which offered support to the view that inoculation with an antigen during the early stages of poliomyelitis infection did increase the incidence of paralysis as well as determining its site; the data, however, gave no guide to the relative risks involved, for example, between the different antigens.[2] Elsewhere, in its "Annotations", *The Lancet*[3] has quoted evidence from a report by Dennis Geffen, based on London data, supporting the association between the site of paralysis and inoculation, but without clear relation to a specific antigen.

Academically, these findings and their discussion are of great interest, though it is clear that much more work must be done before the facts can be explained, and indeed before it is clear what are all the facts. Members of the practising profession, however, especially the general practitioner, are faced with an immediate practical problem, and some clear lead, based on the available knowledge, incomplete though it is, should be given by those best qualified to give it. There seems to be little, if any, reason to withhold reassurance from parents on diphtheria immunization, and it would be tragic to see any decline in the notable successes achieved by public health authorities in this field. Pertussis immunization calls for more caution. The problem involved is possibly bound up with the difficulty, especially for parents, of taking a sane view of poliomyelitis. The general fear of this disease, with its spectacular effects in a quite limited number of cases, is out of all proportion to its real dangers as compared with those of more commonplace epidemic diseases. By contrast, the lethal qualities of whooping cough amongst babies in the first year of life are too rarely appreciated. The efficacy of prophylactic measures against pertussis is, of course, not fully established, or at least is not so great as the efficiency of measures against diphtheria. Public health authorities, probably wisely, have not favoured mass immunization against whooping cough because limited success may adversely affect public opinion on diphtheria immunization. However, authoritative opinion supports the value of pertussis vaccine, and the average practitioner recommends its use to parents. Its degree of protective value, together with the potential dangers of whooping cough according to the age of the child, must now be balanced against the apparently greater risk of paralytic symptoms in the event of infection with the poliomyelitis virus; the present evidence does not indicate increased susceptibility to poliomyelitis infection. Considered thoughts on these aspects by pædiatricians and others suitably qualified would be welcomed.

[1] *The Lancet*, April 29, 1950.

[2] The report on this investigation, by Hill and J. Knowelden (both medical statisticians of high standing), has since appeared in the *British Medical Journal* of July 1, 1950. It supports the influence on incidence and localization of paralysis of inoculation within one month before onset of illness, but not for a longer period. It fails to support the belief that pertussis and combined vaccines are more prone to produce paralysis than others, such as "A.P.T.", though this is not conclusive.

[3] *The Lancet*, April 15, 1950.

CHILBLAINS.

In the past measures for the treatment of chilblains have been characterized by multiplicity, variety and ingenuity rather than by efficacy. Some of these measures were listed in these columns on January 25, 1947, but their reiteration is unnecessary; they were alike unsatisfactory whether their origin was medical or popular. However, two rather more promising remedies have come to light since then. Most of our readers will be familiar with an article by R. J. Gourlay,[1] in which he described gratifying results from administration of nicotinic acid. His dosage was 50 milligrammes for an adult (25 milligrammes for a child) given three times a day after meals; this was increased in severe cases, but more than 300 milligrammes a day was never required. Continued administration was often needed; in some cases relapse followed withdrawal of the treatment, but its resumption brought immediate relief.

An interesting report on the experimental and clinical effects of vitamin *K* on chilblains has now been published by David Wheatley,[2] following up a preliminary report which attracted some attention in 1947. Wheatley's patients numbered 26. The prothrombin times of 16 of these were estimated, but in only one case was there a slight deviation from normal in the acute stage of chilblains. In one case the prothrombin time was estimated in capillary blood from the chilblain itself, but this again was normal. There thus appears to be no support for theories of the cause of chilblains based on prothrombin deficiency. Clinically 23 patients responded completely to treatment with vitamin *K*; three failed to respond, and the failure persisted despite the addition of various measures to improve capillary circulation in the chilblains. The dosage of vitamin *K* was 20 milligrammes given three times a day by mouth. As with nicotinic acid, continued administration appears to have been necessary; relapse followed cessation of therapy, with relief on resumption. Thus the therapeutic results were quite striking, though no experimental evidence was obtained in support of the therapy. No doubt these two forms of treatment will have wide testing and more conclusive assessment will be possible. Wheatley remarks that the number of patients studied in his series is small because the public has lost faith in the medical profession's ability to treat chilblains and comparatively few people with chilblains consult a doctor. Perhaps the introduction of these apparently useful agents will change this. It is certainly undesirable that a substance of the type of vitamin *K* should be used for self-medication.

[1] *British Medical Journal*, February 21, 1948.

[2] *The Lancet*, April 15, 1950.

Abstracts from Medical Literature.

RADIOLOGY.

Carcinoma of the Thyreoid in Children.

HUGH F. HARE AND RICHARD V. NEWCOMB (*Radiology*, March, 1950) have reviewed five cases of thyreoid cancer in children followed for from thirteen to eighteen years. They state that these five patients with thyreoid cancer have shown a few points in common, the most important being the long survival time; this corresponds to the experience of other writers on this subject. Another interesting and important fact is that pulmonary metastases of a similar appearance developed in two of the patients many years after the removal of the primary tumour. The two patients with metastases to the lungs had different pathological pictures. In both cases, however, the cancer had arisen in lateral aberrant thyreoid tissue, which may be of some significance. In two cases, after the original tumour had apparently been cured, multiple colloid adenomatous goitres developed, possibly on a compensatory basis. Radiation therapy is indicated as an adjuvant to operation in the treatment of all carcinomata of the thyreoid. The authors' cases illustrate the value of this procedure, since in three of the four cases in which irradiation was used, there was regression of the recurrent or metastatic lesion. The authors make the following conclusions: (i) that any nodular mass in the region of the thyreoid in children should be suspected of being malignant until proved benign by excision and biopsy; (ii) that X-ray therapy is a most important adjuvant to surgery in the treatment of thyreoid cancer; (iii) that distant metastases in carcinoma of the thyreoid do not necessarily portend an unhappy outcome.

X-Ray Examination in Hypertrophic Pyloric Stenosis.

HANS W. HEFKE (*Radiology*, December, 1949) considers that in cases of hypertrophic pyloric stenosis, X-ray examination is superior to clinical examination and should be used more extensively, especially in early or doubtful cases. No preparation is necessary, but it is best to withhold feeding for three to four hours. A gavage tube is introduced into the stomach in order to remove food, secretion and gas, and two ounces of a thin barium mixture are injected through the tube. Fluoroscopic examination is not employed and is at present thought unnecessary. The anatomical structures involved are so small that actual fluoroscopic observation is not reliable. Furthermore, repeated fluoroscopy exposes the infant to more radiation than is advisable. The diagnosis is made from films, of which the first are taken five to ten minutes after administration of the barium. Because the routine antero-posterior or postero-anterior film of the stomach fails to depict the pylorus and duodenal cap in infants, in more than 50% of cases, only films in the right anterior oblique position are used. Occasionally a right lateral film is added. The time interval between the introduction of barium into the stomach and the first films is of importance. Normally the barium begins to leave the stomach immediately, or not later than after a period of five or ten minutes, which is called the pyloric opening time. The author has not observed a longer pyloric opening time than ten minutes in several hundred normal infants or in infants with pyloric spasm or other conditions causing vomiting. When, more than ten minutes after the barium was given, none has left the stomach, the diagnosis of pyloric stenosis becomes very likely. In that event, two more right oblique films are taken thirty minutes after barium administration. Such abnormal prolongation is a definite sign of hypertrophic pyloric stenosis. The most reliable radiological sign is the demonstration of the narrowed elongated pyloric canal, the so-called "pyloric string sign". It is observed as a thin streak of barium, 1·5 to 2·5 centimetres long, in the prepyloric region of the stomach. While it can at times be seen on routine postero-anterior films, it shows to best advantage on right oblique views. It has not been observed in normal infants or in infants with other diseases. It is present in almost two-thirds of the cases on the first set of films, that is, five to ten minutes after the barium is given. When, however, the pyloric opening time is considerably delayed, the string sign may not be seen until the thirty-minute films have been taken, or even later, depending on the time when some barium leaves the stomach.

The Osseous Lesions of Sarcoidosis.

JOHN F. HOLT AND W. I. OWENS (*Radiology*, July, 1949) state that the medullary cavity is the primary seat of the epithelioid tubercles of sarcoidosis and that frequently these medullary lesions are much more extensive than radiological examination will indicate. Usually, if sarcoid is present in the marrow, secondary changes in adjacent bone occur in one of several patterns. If progress of the granulomatous process is slow and if lacunar resorption takes place in a fairly uniform manner over a relatively large area (for example, an entire phalanx), diffuse enlargement of the lacunæ occurs, resulting in mottled rarefaction of the bone. This can be recognized radiographically in its early stages as a stippled pattern, with tiny dots of diminished density projected against a background of bone of normal or near normal density. There is a definite tendency for the rarefaction to develop more intensely at certain points, such as the distal ends of the proximal and middle phalanges and the proximal ends of the distal phalanges. Here the lacunæ not only enlarge, but also coalesce to produce localized cavities of varying sizes and shapes. In addition to the changes which develop secondary to medullary foci, perivascular infiltration of the Haversian canals may occur, with resultant thinning of the cortex and destruction of the finer trabeculæ. In the very early stages of this pathological process, the X-ray film shows apparent localized osteoporosis, which is entirely non-specific and hence not characteristic. Later this non-specific deossification is supplanted by a reticular pattern of bone destruction, which appears to be the most common and most specific single X-ray manifestation of osseous sarcoid. Two additional very important aspects of sarcoid lesions in bone are the relative inviolability of the periosteum and the almost total absence of joint involvement. Radiologically, the absence of periosteal thickening in bone sarcoid is one of the most valuable of all diagnostic signs. In fact, so reliable is it that when evidence of periostitis is present one should seriously question the diagnosis of sarcoidosis. The same is true of bone sequestra, which with their accompanying draining sinuses are almost never seen in this disease. The absence of joint involvement is equally striking, and patients may maintain normal or nearly normal articular function even when severe mutilating deformities are present.

Hamartoma of the Lung.

E. A. BRAGG AND G. LEVENE (*Radiology*, February, 1950) state that hamartoma of the lung is probably second only to bronchogenic carcinoma in occurrence. In the past, the majority of cases of hamartoma of the lung have been classified as chondromata because of the predominance of cartilage. It is probable that some cases in which the glandular elements predominate have been erroneously diagnosed as bronchial adenoma. Others may have been called dermoids, but the absence of any tissues not found normally in the lung should serve to distinguish a hamartoma from a true dermoid tumour. The X-ray diagnosis of hamartoma is not always possible and rests heavily upon the presence of calcification within the tumour. The usual findings are as follows: (i) a sharply defined tumour in the lung parenchyma with clear lung tissue surrounding it; (ii) lobulation of the margins in most cases; (iii) peripheral or subpleural location in most cases; (iv) irregular patches of calcification in many cases; (v) areas of lesser density near the periphery of the tumour in some cases, due to collections of fat. As benign tumours in which malignant degeneration is extremely rare, hamartomata of the lung will never be of great importance surgically in their own right. They are important in that current practice calls for exploration of all discrete pulmonary tumours other than those known to be of metastatic origin. With the rising frequency of discovery of asymptomatic tumours in mass chest surveys, the incidence of surgical cases of hamartoma of the lung will increase sharply. By careful study, with laminagrams if necessary, it will be possible to identify a high percentage of these tumours before operation. It is probable that from time to time the radiologist can be of great service in diagnosing a hamartoma in a patient who, because of age or illness, would be a poor or unacceptable "risk" for surgery. In these circumstances a useless operation might be avoided and fear of malignant disease be eliminated.

Early X-Ray Changes in Idiopathic Ulcerative Colitis.

TOUFIC H. KALIL AND LAURENCE L. ROBBINS (*Radiology*, July, 1949) state that the early gross pathological changes of ulcerative colitis include œdema and swelling of the mucosa accompanied by very small ulcers. Radiologically the first corresponding change suggesting the diagnosis is a

difference in the appearance of the mucosal folds; they seem coarser and tend to become parallel, in contrast to their normal irregularity of pattern. These changes, demonstrable on the post-evacuation X-ray film, are not pathognomonic of ulcerative colitis, since any irritative process, such as repeated catharsis, may produce enlarged mucosal folds. In true ulcerative colitis, however, the initial abnormalities are followed by superficial ulceration of the involved lymphoid follicles. On direct visualization, these appear as pinpoint to pinhead erosions, producing the familiar pale, granular, easily bleeding mucosa. For accurate X-ray diagnosis, therefore, demonstration of these shallow tiny ulcers is necessary. In the well-prepared bowel, barium filling at this early stage will show, on close inspection, scattered minute serrations in the involved areas, often seen best in profile in the sigmoid and transverse colon, since these segments lie nearest the film in the customary postero-anterior projection. As the disease progresses, serial X-ray pictures reveal extension of the ulcerative process, producing multiple instead of occasional serrations, so that the entire colon, including the rectum and sigmoid, will show involvement of varying degrees in films of good quality taken at a speed fast enough to offset intraabdominal movement. Care must be taken in distinguishing between these tiny serrations and small irregular defects caused by particles of retained fæces adherent to the bowel wall. On the post-evacuation film the serrations may persist; they are best noted if emptying has been incomplete. This fact suggests that overfilling of the bowel with barium early in the disease can stretch the mucosa sufficiently to efface the minute irregularities, but coats the bases of the ulcerations, so that after partial emptying the irregularities become evident. On the post-evacuation film, also, the mucosal folds appear much thickened and diminished in number; often in the severely involved portions longitudinal folds only are evident.

PHYSICAL THERAPY.

Hodgkin's Disease.

M. V. PETERS (*American Journal of Roentgenology*, March, 1950) surveys patients with Hodgkin's disease treated between 1924 and 1942 in the Ontario Institute of Radiotherapy at the Toronto General Hospital. Only patients whose original pathological pictures have been reviewed and verified are included. Half of the entire group of patients gave a history of having first noted enlargement of one of the cervical lymph glands. Some patients who were treated for suspected Hodgkin's disease of the mediastinum and who are still alive have not been included since the diagnosis is necessarily unproved. Between 1924 and 1942 inclusive, a total of 257 patients with a diagnosis of Hodgkin's disease were treated by irradiation. The overall five-year survival rate of this group was 38%. One group of 60 patients with no histopathological verification of the diagnosis, but having a five-year survival rate of 33%, and a second group of 84 patients having a five-year survival rate of 22%, but with incorrect pathological diagnosis, were excluded. The remaining 113 patients with pathologically proved and verified Hodgkin's disease had a gross over-all five-year survival rate of 51% and a ten-year survival rate of 35%. It is stated that these survival rates are considerably better than any others reported in the literature to date. It is noted that in early cases with involvement of a single lymph-gland region or a single lesion elsewhere in the body, the survival rate is consistently high at the end of both five and ten years. In cases with multiple lymph-gland involvement on the patient's admission to hospital, the survival rate is consistently poor. The presence of constitutional symptoms, either as a first complaint or during the course of the disease, also has a definite influence on the prognosis. Among patients with no constitutional symptoms at any time, the survival rates in 40 cases at the end of five years and ten years were 85% and 65% respectively. Among 36 patients with constitutional symptoms, but not as the first complaint, the survival rates at the same periods were 36% and 8%; and among 37 patients with constitutional symptoms as the first complaint, the figures are 30% and 11%. In women the disease tends to run a more chronic course than in men. It is concluded from the survey that the chief factors in the control of Hodgkin's disease are early diagnosis and a more optimistic attitude toward prognosis in early cases, early institution of radiation therapy, with moderately intensive treatment in hopeful cases, frequent follow-up and early treatment of any patients with recurrences.

X-Ray Therapy and the Relief of Pain.

F. W. O'BRIEN (*Radiology*, January, 1950) states that most authors in discussing the properties of X rays make scant mention of the analgesic effect. He discusses the value of X-ray therapy in relieving pain in such conditions as boils and carbuncles, in calcification of the supraspinatus tendon or so-called subacromial bursitis, in *herpes zoster*, in Marie-Strümpell's spondylitis and in metastatic bone lesions. In 60 cases of carbuncles in which treatment was by X-ray therapy alone, the pain was relieved in from three to nine hours after the first treatment in every case. The author states that the relief of pain is explained by the relief of tension in the tissues as the inflammatory process is resolved. A similar explanation may fairly be given for the dramatic relief of pain often seen in cases of subacromial bursitis following X-ray therapy. It is not an uncommon experience to see a patient with an arm in a sling, sleepless because of pain, appear a day after X-ray therapy and report freedom from pain and a restful night's sleep. X-ray therapy has been shown to inhibit exudation and decrease the number of inflammatory cells. In *herpes zoster*, the irradiation is directed to the region of the spinal nerve roots believed to be involved, and in most cases relief of pain is obtained. X-ray therapy is accepted now as probably the most satisfactory method of treatment in Marie-Strümpell's spondylitis. Relief of pain is obtained in most cases and often the disease may be arrested. In metastatic bone lesions, pain is frequently relieved by X-ray therapy. It is emphasized that a patient complaining of skeletal pain following an operation for a primary metastasizing neoplasm should have deep X-ray therapy even though X-ray films show no abnormality. It is suggested by the author that the relief of pain in Marie-Strümpell's disease, *herpes zoster* and metastatic lesions of the spine is due to its effect on the autonomic system.

Röntgen Therapy Alone in the Treatment of Advanced Cervico-Uterine Cancer.

F. BACLESSE (*American Journal of Roentgenology*, February, 1950), of the Curie Foundation, Paris, reports on 78 patients with advanced uterine cancer treated by deep X-ray therapy alone from 1930 to 1942. Of 72 patients with cancer in stages III and IV irradiated with 200 kilovolt X rays, 14 are alive and apparently well five years or more after treatment. Of six patients treated with 500 kilovolt X rays, there are no survivals; but these figures are inconclusive because the number of patients is too small and the technical conditions have not been identical. Of the 78 patients treated, seven had much involvement of the vagina, extending to the vulva, and of these, four remain apparently cured after five years. Careful attention has been given to strict clinical classification of the parametrial involvement, and apparently a few patients remained cured despite true involvement of the parametrium. The author states that the tendency in later years has been to use smaller fields of treatment and increased fractionation up to eight or ten weeks or even twelve weeks, so as to avoid cutaneous, intestinal or vesical complications. The tumour dose for the cured patients has been from 4300*r* to 7100*r*. As well as the usual anterior and posterior oblique pelvic fields, two small perineal fields (five by six centimetres) directed towards the *obturator foramen* have been used. It may be possible in radiosensitive cases to deliver an adequate dose of 5000*r* or more to sterilize the parametrium.

MEDICINE.

Multiple Sclerosis and the Vegetative Nervous System.

LEO HESS (*The Journal of Nervous and Mental Disease*, January, 1950) discusses multiple sclerosis (*encephalomyelitis disseminata periaxialis*) and the vegetative nervous system. He states that a review of the literature shows appreciation by many authors of "crises" of the stomach. He records hyperacidity and hypermotility of the stomach in every patient in a group of 23 cases. Hyperperistalsis occurred throughout the entire process of gastric digestion; peristaltic waves originating high in the fundus extended quickly to the pylorus. It is surmised that there is increased vagal activity. The hyperacidity and spasm of the stomach are not associated with subjective symptoms. The author believes that this syndrome has a central nervous origin. He notes that freedom from pain likewise occurs in patients who have undergone lobotomy.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association, in conjunction with the Pharmaceutical Society of New South Wales, was held at the Robert H. Todd Assembly Hall, British Medical Association House, 135 Macquarie Street, Sydney, on April 27, 1950, DR. G. C. HALLIDAY, the President of the Branch, in the chair.

The British Pharmacopœia.

PROFESSOR R. H. THORP read a paper entitled "The British Pharmacopœia from the Viewpoint of the Pharmacologist" (see page 215).

MR. K. L. PETHERBRIDGE (Pharmaceutical Society), in opening the discussion, said that viewed from the angle of the pharmacist, the chief features of the 1948 British Pharmacopœia were, firstly, the increased representation of drugs by their active ingredients rather than as galenical preparations, and secondly, the large number of specific therapeutic agents included chiefly in tablet or injection form. The trend seemed therefore to transfer the art of pharmacy from the practising pharmacist to the manufacturing pharmacist, as the conditions of preparation and presentation of many of the modern drugs demanded greater facilities than it was possible for the practising pharmacist to provide. That did not necessarily signify that pharmacists were to be displaced from their traditional role. The British Pharmacopœia had in many cases ensured that it was possible for the pharmacist to keep himself abreast of current trends, a case in point being the methods of sterilization, which, as Professor Thorp had already mentioned, had been compiled with a view to pharmacy conditions. The advent of those new techniques, included in the fourth addendum of the 1932 British Pharmacopœia and continued in the present issue, brought sterilization more within the reach of the practising pharmacist than ever before. Of the four official methods of sterilization, pharmacists could readily undertake three: sterilization by heating in an autoclave, sterilization by heating with a bactericide, and sterilization by dry heat at 150° C. The fourth official method, filtration, with its accompanying tests for sterility, was beyond their scope. The provision of the official non-pyrogenic water for injection, for use in the bactericide sterilization process, was for pharmacists the major change in the latest sterilization methods. That would necessitate a special still fitted with a splash trap. The point raised by Professor Thorp regarding the certainty of the temperature of the autoclave would doubtless reassure those pharmacists who used constant boiling mixtures to obtain 115° C. for their sterilization. It would appear that such a bath with a thermometer immersed therein was likely to be more efficient in the hands of the occasional operator than an autoclave.

Mr. Petherbridge went on to say that because of more frequent use, more interesting changes in the British Pharmacopœia were found in the newer ointment bases, and more particularly in the use of the new emulsifying agents. Wool alcohols and the water-in-oil emulsions produced therefrom were little different from wool-fat and its preparations, but the oil-in-water emulsion bases were a definite innovation. The penicillin creams were the prototype of a series of cosmetically elegant creams. Wider mention of their possibilities was made in the monographs of the "Australian Pharmaceutical Formulary", which included a list of drugs suitable for incorporation in that type of base. The increased absorptive properties of such creams was recognized by a warning that careful consideration should be given to the strengths of active ingredients. Those creams presented the first successful break from the limits of the anhydrous paraffin, or the wool-fat water-in-oil emulsion bases; and it was pleasing to see pharmacopœial recognition given so quickly to emulsifying wax as the emulsifying agent responsible—a step certainly due to the penicillin cream base. With reference to penicillin cream it was noted that the British Pharmacopœia monograph specified that the cream was to be freshly prepared as it lost potency on storage. From those considerations it was obvious that penicillin cream could conform to standards only when it was extemporaneously prepared by the pharmacist, and the strength of manufactured samples must raise grave doubts. It was felt that specified conditions of storage and a time limit for use would be a welcome addition to the monograph. Apart from the creams, penicillin preparations which had apparently been formulated with the pharmacist in mind, were the ointment, the oculentum and to a lesser extent, the oily injection. The need to use peroxide-free oil in the preparation of the last mentioned put it rather beyond the scope of the pharmacist without facilities for such chemical evaluation. Such non-official preparations as eye drops, dusting powders, suppositories, pastilles and mixtures were everyday tasks, and by study of the methods of preparation of official products, suitable techniques might be devised.

Mr. Petherbridge said that as a book of standards the British Pharmacopœia stood absolute, but as a book of reference it was marked by some omissions. Professor Thorp had mentioned that the British Pharmacopœia doses were intended to form a useful guide to the physician, but were in no way binding and, in fact, were usually conservative. From the pharmaceutical viewpoint, that was unfortunate. To a certain degree final responsibility for dosage still fell upon the pharmacist. The "Australian Pharmaceutical Formulary" stated that when it was impossible to get into contact with the prescriber and in the absence of initialling of an unusually large dose, the pharmacist must reduce the dose to one that he considered was within the limits of safety. That procedure had presented no great problem until recent years, as the properties of drugs in use were comparatively well known. With the tremendous strides in medicine during the last fifteen years and the advent of many new drugs, standard works of reference lagged far behind established usage. Thus when a pharmacist was confronted with a large dose of any drug issued in the last two to three years, he had no means of checking whether such a dose might be toxic or otherwise. The position became more difficult with doses for young children, and deciding whether a British Pharmacopœia overdose, as calculated by Young's rule, of such a drug as pethidine was dangerous then became a major task. Something was required to fill the gap between the introduction of a new drug and the reports of its normal dosage, such as appeared in Martindale's "Extra Pharmacopœia". Whilst it was probably beyond the scope of the British Pharmacopœia to define strict limits of dosage, and indeed undesirable that it should do so, consideration could be given to the introduction of suitable notes regarding tolerance to new drugs, especially in children.

Mr. Petherbridge considered that pharmacists would concur with the renewed suggestion to abandon the apothecaries' system of weights and measures in favour of the scientific metric system, but a tremendous amount of replacement would be necessary before it could become an accomplished fact. Not the least of the changes would be the production of suitable containers for liquid preparations, graduated to take advantage of the decimal system, with doses being set out in multiples of ten. A further anomaly was the fact that in New South Wales metric weights and measures were unstamped and as such were illegal.

In conclusion, Mr. Petherbridge said that the pharmacist would be happy to give complete support to any efforts which might reduce the multiplicity of trade names appearing for the same compounds. Agreement on any complete remedy for the situation appeared to be remote, but the pharmacopœial issue of approved names for proprietary substances marked the first step of any importance in the campaign; and it was to be hoped that it would provide sufficient stimulus to bring about a thorough attack on the problem.

DR. F. HALES WILSON said that Professor Thorp had given an interesting and useful address on a somewhat forbidding subject, and the size of the large audience was an indication of its importance and of the interest it aroused. Dr. Wilson pointed out that the standards of the British Pharmacopœia were essential to medical practice. However, so far as the average doctor was concerned, the British Pharmacopœia was like the foundations of some great edifice, very important but never seen. In preparation for the meeting Dr. Wilson had recently visited one of the local hospital libraries and sought to borrow the book. The librarian was loath to lend such an important work of reference, and he was sufficiently impertinent to ask her how often it was used. She replied: "On rare occasions by the laboratory staff and never by the honorary medical officers."

Dr. Wilson went on to say that the 1948 British Pharmacopœia contained many important additions; some of the deletions were also interesting, such as buchu, guaiacol, hexamine, jalap, lobelia, sulphapyridine ("M & B 693"). Ammonium carbonate was dropped, but ammonium bicarbonate, which was more stable and therapeutically equally valueless, remained. Dr. Wilson said that several portions of Professor Thorp's paper were of special importance. He (Dr. Wilson) referred to the preparation of pyrogen-free solutions and to the necessity of using only single-dose containers for substances to be injected intrathecally. The same rule should apply to the preparation of

penicillin solution for intrathecal injection, and to the local anæsthetic used before such injections. Those specially interested in diabetes would be intrigued by the remarks about insulin and its standardization. Regarding protamine zinc insulin, apparently the protamine content was constant for any one manufacturer's product, so that mixtures of soluble and protamine zinc insulin worked very well in practice, but one might have to vary the rates when changing to a different brand. Dr. Wilson considered that Professor Thorp's remarks about trade names were very timely. The bewildering list of aliases prevented a busy practitioner from keeping abreast of new substances. The compilers of the British Pharmacopœia would not use a proprietary name and preferred one which indicated the chemical composition of the substance. Therefore, when a new preparation had established its claim as a useful therapeutic addition, it was re-christened before appearing in the British Pharmacopœia. They had first learned to know an extremely useful mercurial diuretic by the name "Salyrgan"; in the British Pharmacopœia that substance was mersalyl, and another trade name was "Neptal". A respiratory stimulant, "Coramine", was now official by the name nikethamide and called "Anacardone" by one manufacturer. Dilantin was officially known as phenytoin; phemitone, formerly the British Pharmacopœia name of "Prominal", had fortunately been changed to methyl phenobarbitone. It would be not unreasonable to ask that the British Pharmacopœia name be shown on the label in type as large as or larger than the proprietary name. The introduction of approved names for new substances which, if they proved their worth, would later be included in the British Pharmacopœia without change of name, was an excellent step.

In conclusion, Dr. Wilson said that some method whereby busy practitioners could be kept in touch with progress in therapeutic substances was very desirable. Perhaps Professor Thorp would issue a brief annual review in THE MEDICAL JOURNAL OF AUSTRALIA. If he (Dr. Wilson) was a more frivolous person, he would suggest an even better way: the British Medical Association should send them the information attached to suitable pieces of blotting paper. The present deluge of that form of literature was such that one might almost be pardoned for interpreting the letters "B.P." as referring to the blotting paper from which the doctor obtained his information.

MR. A. G. SHORT asked what was to be done about standard preparations which had not been included in the 1948 British Pharmacopœia.

Professor Thorp replied that new drugs would be indicated in addenda and older ones in the British Pharmaceutical Codex. Pharmacists should adhere to the order of the prescribing physician. When a preparation was omitted from the British Pharmacopœia, it was an indication that the Pharmaceutical Committee had weighed all the evidence for and against a preparation and had concluded that it had no useful place in the pharmacopœia. At the same time, some physicians might have found that the preparation which had been omitted was of use in their experience. In such circumstances the physician should indicate on his prescription the year in which the preparation had been listed.

MR. H. W. READ referred to ammonium carbonate. He said that the British Pharmacopœia stated that when ammonium carbonate was ordered, ammonium bicarbonate should be dispensed. Mr. Read wished to know what a pharmacist should do.

Professor Thorp described the question as a poser. If a physician wanted ammonium carbonate for his patient, he should include the words "B.P. 1932" on his prescription. The trouble was that ammonium carbonate was not a definite substance, but the bicarbonate was.

Mr. Read referred to the problem of what should be done when ammonium carbonate was included in an old prescription.

Professor Thorp replied that legally the pharmacist should use the bicarbonate as laid down in the British Pharmacopœia of 1948. He thought that if an old prescription was presented, the pharmacist should use his judgement.

MR. A. T. CAMPBELL referred to the preparation of penicillin cream, and asked whether aseptic precautions should be used in its preparation and dispensing.

Mr. Petherbridge replied that special aseptic precautions were not essential, but were desirable. Anything which would tend to exclude the introduction of penicillin-resistant bacteria should be done.

MR. J. MORRIS asked what was the length of time for which penicillin ointment and creams, particularly proprietary brands, could be used after storage.

Professor Thorp replied that much work had been done on this subject by manufacturers, and he thought that if such preparations were carefully stored in a refrigerator, they would be reasonably active after a period of some months, although this would vary considerably from one preparation to another.

MR. E. B. POLLARD referred to the different dates on which the British Pharmacopœia, 1948, was proclaimed in the several Australian States.

MR. A. E. CONNOLLY, the secretary of the Pharmaceutical Association, replied that there was no Act in New South Wales under which the date for use of a new edition of the pharmacopœia could be proclaimed. A round-about method was adopted. The new edition of the pharmacopœia was gazetted as having arrived, and that was regarded as sufficient. Another edition of the British Pharmacopœia might be expected in 1952.

MR. B. M. DASH asked Professor Thorp whether he shared the opinion on the obsolete nature of sulphapyridine.

Professor Thorp agreed that the newer sulphonamides were much superior to that drug.

The meeting concluded with a vote of thanks to the speakers, proposed by Mr. Read.

Correspondence.

AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

SIR: It has been my privilege—and enjoyment—to attend the last three sessions of the Australasian Medical Congress.

It is possibly inevitable that a medical congress should in large measure be devoted to expert discussion between specialists. The general practitioner admittedly is given the opportunity to attend any session he so desires, thereby enriching his knowledge of the particular speciality concerned. It is also presumably inevitable that there cannot be a "general practitioners" section of Congress where those who in number comprise the majority of the profession could discuss their mutual problems, perhaps with the valued help of appropriate specialists.

"Combined meetings" are a welcome feature of Congress, consisting as they do of the assembling of two or three or more groups of specialists met to discuss signs, symptoms and diseases of common interest, whether the general subject be "headaches" or "tuberculosis". At the Congress recently assembled in Brisbane, the single plenary session particularly was a first-rate meeting, giving pleasure and profit to all attending. May I suggest, sir, through the courtesy of your columns, that consideration be given to the holding of more than one plenary session at the next Australasian Medical Congress? Should these sessions be conducted on the same happy lines as that of the Brisbane Congress and their chairmen have the same gifts, great pleasure and profit would be undoubtedly given to many.

Yours, etc.,

K.B.

Sydney,
July 4, 1950.

RHEUMATISM AND THE CONGRESS.

SIR: I must congratulate the committee of experts for the excellence of their answers on questions of infantile and cardiac rheumatism and rheumatic fever. However, in rheumatoid disease many who listened carefully were astounded at some of the expressed opinions, and these cannot be allowed to go unchallenged.

Those of us who treat many rheumatoid arthritics know how individual is each case and how each must be investigated and treated on its merits. To state that no "expensive" laboratory investigations are necessary is misleading. All rheumatoids, except "burnt-out" cases, require some laboratory investigation, which, fortunately, is not an expensive item in Queensland. Surely here is a disease that should not be treated in a perfunctory manner without full and proper investigation.

To decry the removal of septic foci surely was not meant by the committee to include what has been designated "likely" foci (Fletcher, 1947). The indiscriminate removal of foci obviously deserves censure. Almost every recognized authority (Copeman, 1948; Fletcher, 1947; Steinbrocker, 1942) on rheumatoid disease has advocated the removal of septic foci when, as is often the case, there is some sound reason for doing so. And how, may I ask, can that essential in treatment, the improvement in general health, be attained and maintained in the presence of chronic toxæmia from a focus?

To proclaim antibiotics including penicillin as having "no place in the treatment of rheumatoid disease" and of being "often harmful" is contrary to the opinion and practice of recognized authorities (Copeman, 1948). Penicillin is the best preventive of a flare-up of polyarthritis following necessary surgical and dental procedures in rheumatoid arthritics.

The statement that "gold should be dropped" is rather premature in the present state of our knowledge. Only the indiscriminate use of gold should be deprecated. Gold can be one of the most useful adjuncts to treatment; but one must be thoroughly conversant with the types of cases: (i) who must not have gold; (ii) who can be given gold with certainty of great benefit; (iii) who, if given gold, will obtain no effect as regards their arthritic condition. It was disappointing that indications, contraindications, dosage dangers *et cetera* for the use of gold did not receive adequate ventilation. As regards the use of gold, American authorities state that rheumatologists "with few exceptions, report results therefrom better than those obtained with any other single remedy" (Hench *et alii*, 1948).

The hormones ACTH and cortisone, and others with less offensive side effects, are at present unobtainable. More guidance, therefore, could have been given in the use of those remedies which we already have and know (or should know).

Yours, etc.,

149 Wickham Terrace, Brisbane, June 5, 1950.

JOHN A. SHANASY.

References.

Copeman, W. S. C. (1948), "Textbook of Rheumatic Diseases", pages 133, 134.

Fletcher, E. (1947), "Medical Disorders of the Locomotor System", Chapter VII.

Hench, P. S., *et alii* (1948), "Rheumatism and Arthritis: Review of American and English Literature of Recent Years (Ninth Rheumatism Review)", *Annals of Internal Medicine*, Volume XXVIII, page 131.

Steinbrocker, O. (1942), "Arthritis in Modern Practice", page 52.

EYE TESTING FOR DISTANT VISION.

SIR: It is customary to test eyes subjectively for distant vision at six metres. I believe that far more accurate glasses would be prescribed if this distance were greatly increased. I find that the greater the distance for the test, the more correct are the lenses ordered.

For distant use people desire glasses to enable them to see films at picture shows and to see a hundred yards or more ahead when driving an automobile, not only to see across a room.

The six metre test is wrong by both physics and physiology.

Yours, etc.,

105 St. George's Terrace, Perth, July 11, 1950.

F. SIMPSON, D.O. (Oxon.).

SECOND INTERNATIONAL CONGRESS OF CRIMINOLOGY.

SIR: The secretary-general of the congress has asked me to make known that the International Congress of Criminology will be held in Paris on September 10-19, 1950. Amongst others, specialists in psychiatry, psychology and forensic medicine are invited to contribute papers and, of course, attend. I have a few copies of the programme and other information, but anyone who proposes to attend the congress should apply at once to the secretary-general, Monsieur P. Piprot d'Alleaume, 188 Avenue Victor Hugo, Paris, XVI^{e}.

Yours, etc.,

135 Macquarie Street, Sydney, July 18, 1950.

W. S. DAWSON.

INFECTION IN THE MIDWIFERY NURSERY.

SIR: I would be grateful for space to clarify my remarks to the congress reported in the journal for July 15, page 105.

Speaking of neonatal tetanus, I made it clear that I was describing the results obtained by Dr. J. I. Tonge at the Laboratory of Microbiology and Pathology, Brisbane.

The points I had intended to make when speaking of intestinal infections were:

1. The link established from mother to her infant to the neonatal nursery was in our series of neonatal Salmonella infections; I did not generalize from this observation.
2. The babies, we believed, were infected at or soon after birth; we had no evidence to suggest infection *in utero*.
3. This discussion was limited to Salmonella infections; we have had no experience at all with neonatal dysentery.
4. The remarks on dysentery were comment on Dr. Stephen Williams's contribution and were about older babies and young children. We had observed a high rate of inapparent infections with Flexner II in young children, although this organism was definitely pathogenic to some children and to adults. We had also observed that infections in the children did not respond well to the sulphonamides used, although the same drugs were quite effective in Flexner II infections of adults.

Knowing the difficulties of reporting accurately during a somewhat involved discussion, I hope that your reporter at the meeting concerned will forgive this attempt to clarify a statement which I may not have made perfectly clear in the first place.

Yours, etc.,

I. M. MACKERRAS.

Queensland Institute of Medical Research, Brisbane, July 21, 1950.

WALKING STICKS FOR THE BLIND, AGED AND INFIRM.

SIR: In view of the increasing mortality rate in association with modern traffic, I would make the following appeal to my fellow practitioners, many of whom are Rotary Club members.

With the success of the white walking sticks for the blind, and the yellow walking sticks for the aged and infirm scheme (of Rotary origin), which will warn motorists and pedestrians of those bearing such sticks and their disabilities, may I seek the cooperation of motorists and others who will give thought to the white and yellow sticks?

Yours, etc.,

KENNETH ADDISON, President, Bondi Junction Rotary Club.

241 Oxford Street, Bondi Junction, New South Wales. May 15, 1950.

Obituary.

PERCIVAL THOMAS SPOWER CHERRY.

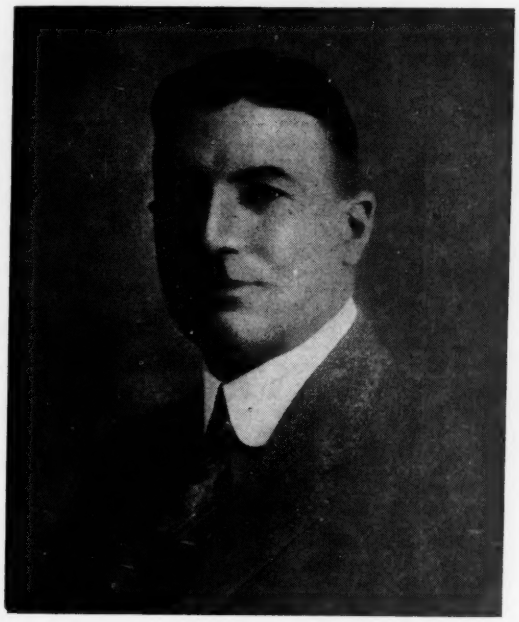
WE are indebted to Dr. F. S. Hone for the following appreciation of the late Dr. Percival Thomas Spower Cherry.

Dr. Percival Thomas Spower Cherry, who died suddenly on April 28, 1950, was born on December 2, 1885, at Wilmington, South Australia, where his father, who was in the South Australian Education Department, was at the time headmaster of the local State school. His boyhood was spent in various country towns and at different State schools, until he went to Saint Peter's College in 1899, having gained a government bursary; he remained at this school until 1902, and then he entered on his medical course at the University of Adelaide. In the meanwhile his father had been moved to become headmaster of the Port Adelaide State school, and except for a few years, the rest of Dr. Cherry's life was spent in the Port Adelaide district; there he closely identified himself with the community life, so that his death has left a gap, both civic and professional, which will take a long time to fill.

After obtaining the degree of M.B., B.S., in 1908, at the University of Adelaide, he was a resident medical officer at the Royal Adelaide Hospital for a year. In 1910 he went into practice for the first time at Tanunda as an assistant to Dr. F. Juttner, with whom he remained until he started practice on his own account in 1912 at Tailem Bend, a railway junction on the river Murray. He was married on August 7, 1912, and of his four children, three have become medical graduates of the University of Adelaide.

In 1915, on the death of Dr. Gething, of Port Adelaide, Cherry bought his practice; he became medical officer to the Port Adelaide Casualty Hospital and quarantine officer for Port Adelaide also. For the next few years I was closely associated with him in his professional and quarantine work, and I learnt to rely implicitly on his interest in his

profession and his thoroughness in attention to his official work. He gradually built up an extensive practice amongst the shipping community at Port Adelaide and closely identified himself with the public life of the Port Adelaide district. In 1922 he was appointed medical officer of health at Port Adelaide, and from that time showed an active interest in municipal and health affairs. He was a representative of the British Medical Association on the board of management of the metropolitan infectious disease hospital when it was first established; he was also largely responsible for introducing an immunization campaign against diphtheria in the district of Port Adelaide. He early became an enthusiastic supporter of the Port Adelaide Football Club, and I have often been told that one of the sights on the Adelaide Oval on a Saturday afternoon was to see the way he threw himself into enthusiastic "barracking" for the local team, of which he was president for a quarter of a century. His quarantine duties kept him from offering himself for active military service, but for years he was a captain on the Australian Army Medical Corps reserve.



For many years he took an active interest in the welfare of the South Australian Branch of the British Medical Association. He became a member of the council in 1924; in 1937 he was made vice-president of the Branch, and he was president in 1938. I have just re-read his presidential address, which was delivered in June, 1939, and have been impressed with the evidence of interest shown there. He remained in office as treasurer of the Branch until his retirement last year. He was a director of the British Medical Association Hall Company. He retained a close connexion with his old school, and was closely associated with the Saint Peter's Old Collegians Association. He was a staunch churchman, and his professional and private life were characterized by his devotion to the highest ideals. He has left behind him fragrant memories in a wide circle of friends, who will miss him greatly, because for a good many years he held, at his home, an annual reunion of those who had graduated at the same time as himself. Remembering him and reading his presidential address, one realizes his pride in remaining a general practitioner rather than striving after academic honours. "An unassuming personality spent in service for others."

Dr. D. R. W. Cowan writes: On April 28, 1950, suddenly and without warning, Percival Thomas Spower Cherry departed this life. No doubt, when our time comes, this is the way most of us would prefer to end this earthly existence. But his passing leaves a gap in his family and professional life that cannot be adequately filled. Only a few days before the end he was looking forward with eager anticipation to a happy time with his wife at the Congress in Brisbane. Percy Cherry typified all that is best in the role of general practitioner and family doctor. Never very enthusiastic to attain for himself the higher degrees of medicine or surgery or to join the ranks of university teachers, yet he left an indelible mark on the practice of medicine and gained a place in the esteem of his professional brethren and of his patients that will not soon be forgotten. This was clearly shown on the day of his funeral by the remarkable array of floral offerings from those desiring to pay tribute to his memory. Percy Cherry graduated in medicine at the University of Adelaide in 1908, and it is of interest to recall that of the nine graduates of that year he was one of four who attained the highest position that the South Australian Branch of the British Medical Association can bestow. After a year as resident medical officer at the Royal Adelaide Hospital he went to a country practice for some years before he started his work in the Port Adelaide district. There he took a leading part in the life of the community and remained in active practice until the day of his death. He was medical officer of health for very many years, and president of the Port Adelaide Football Club.

Not yet sixty-five years old, he was too young to die. He was greatly needed, and he will be sorely missed in the district in which he laboured so long and so successfully. Despite his busy general practice, he found time for service in the interests of his profession. He occupied with distinction the position of president of the Branch and was its honorary treasurer for several years. His enthusiasm and sound judgement were a real asset to the Council through many troublous times. Not the least of his achievements was to raise a family of which three members followed in his footsteps and became doctors. There is every reason to believe that they will emulate their father's example and maintain the standard he has set so high. Stevenson's words might well apply to him: "There are men and classes of men that stand above the common herd." Percy Cherry was one of these, and the greatest sympathy goes out to his widow and family in their grievous loss.

ALFRED MEILLON LANGAN.

We regret to announce the death of Dr. Alfred Meillon Langan, which occurred on July 20, 1950, at Cairns.

LESLIE THOMSON GILLESPIE.

We regret to announce the death of Dr. Leslie Thomson Gillespie, which occurred on July 23, 1950, at Perth.

Naval, Military and Air Force.

APPOINTMENTS.

The undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Numbers 36 and 38, of June 29 and July 6, 1950.

Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

Extension of Services.—The services of Surgeon Commander (D.) John Ellis Richards, O.B.E., are extended for a period of one year from 26th April, 1950, under Section 17 of the *Naval Defence Act*.

Retired List.

Termination of Appointment.—The appointment of Surgeon Commander James Mann Henderson, M.C., for temporary service is terminated, dated 31st January, 1950.

Australian Military Forces.

Royal Australian Army Medical Corps.

To be Colonel, 12th April, 1950.—NX73138 Lieutenant-Colonel (Temporary Colonel) A. M. McIntosh.

VFX700115 Honorary Captain M. Howson is appointed from the Reserve of Officers, and to be Captain, 14th April, 1950.

Citizen Military Forces.

Northern Command: First Military District.

Royal Australian Army Medical Corps (Medical).—1/55108 Major D. C. C. Sword is appointed from the Reserve of Officers and borne supernumerary to the authorized establishment of Majors with pay and allowances of Captain (at his own request), 4th May, 1950.

Eastern Command: Second Military District.

Royal Australian Army Medical Corps (Medical): To be Colonel, 12th April, 1950.—2/50001 Lieutenant-Colonel (Temporary Colonel) S. H. Lovell.

Southern Command: Third Military District.

Royal Australian Army Medical Corps (Medical): To be Colonels, 12th April, 1950.—Lieutenant-Colonels (Temporary Colonels) 3/50056 J. G. G. White, O.B.E., E.D., 3/51002 W. W. Lempriere, D.S.O., and 3/50054 E. V. Keogh, D.C.M., M.M.

Central Command: Fourth Military District.

Royal Australian Army Medical Corps (Medical): To be Colonel, 12th April, 1950.—4/35214 Lieutenant-Colonel (Honorary Colonel) J. M. Dwyer, E.D.

Western Command: Fifth Military District.

Royal Australian Army Medical Corps (Medical): To be Colonel, 12th April, 1950.—5/32054 Lieutenant-Colonel (Temporary Colonel) J. H. Stubbe, E.D. The provisional appointment of 5/20781 Captain C. V. Love is terminated, 1st April, 1950.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps.

1st Military District.—The following officers are placed upon the Retired List (1st Military District) with permission to retain their rank and wear the prescribed uniform, 2nd May, 1950: Lieutenant-Colonels A. W. L. Row, A. V. Meehan and S. A. McDonnell, Majors R. F. Craig, D.S.O., M. G. Sutton, H. Flecker, P. A. Earnshaw, K. R. McGregor and R. Grant, Captain (Honorary Lieutenant-Colonel) A. P. Murphy, M.C., Captains (Honorary Majors) W. Crosse and T. M. Mansfield and Captains G. C. W. Holmes and T. L. Cooney. Captain (Honorary Lieutenant-Colonel) Sir R. W. Cilento is retired, 2nd May, 1950.

2nd Military District.—The following officers are placed upon the Retired List (2nd Military District) with permission to retain their rank and wear the prescribed uniform, 4th May, 1950: Lieutenant-Colonels (Honorary Colonels) A. L. Buchanan, V.D., N. D. Dunbar, C.B.E., E.D., A. S. Walker, G. F. Hill and W. Evans, M.C., Lieutenant-Colonels Sir C. B. Blackburn, O.B.E., J. R. M. Beith, D.S.O., W. S. Dawson, A. C. Thomas, D. R. Brown, M.C., and J. L. Digby, E.D., Major (Honorary Lieutenant-Colonel) V. M. Coppleson, Majors N. M. A. Alexander, J. A. Murphy, R. J. Taylor, A. M. Davidson, O.B.E., J. W. Farrer, O.B.E., H. Rayson, M.C., F. Tooth, C. Anderson, M.C., H. M. De Burgh, J. W. V. Hoets, B. B. Blomfield, R. MacQueen, W. A. L. H. Henderson, J. T. Anderson and W. H. Cook, Captains (Honorary Majors) G. A. M. Heydon, M.C., R. J. Silverton, W. F. L. Liggins, F. N. Rodda, L. May, F. Goldschlag and C. R. Wiburd, Captains E. P. Holland, C. G. Templeman, J. Wall, C. R. Alexander, V. A. Conlon, J. J. Donnellan and A. W. Dean, and Honorary Captain H. E. Thomas. The following officers are retired, 4th May, 1950: Honorary Lieutenant-Colonel B. T. Edye, Honorary Major B. G. Wade and Honorary Captains B. W. Stevenson and N. W. Hansard.

3rd Military District.—The following officers are placed upon the Retired List (3rd Military District) with permission to retain their rank and wear the prescribed uniform, 4th May, 1950: Lieutenant-Colonels (Honorary Colonels) F. E. Keane, M.C., E.D., C. W. B. Littlejohn, C.B.E., M.C., and J. E. Gillespie, E.D., Honorary Colonel F. H. Moran, M.C., Lieutenant-Colonel J. B. D. Galbraith, Majors R. D. Bartram, M.C., M. H. Mailer, M.C., S. C. Fitzpatrick, M.C., A. F. Maclure, O.B.E., A. P. Drummond, W. L. Armstrong, D. D. Browne, V. C. Brown, M.C., T. D. Freeman, A. J. Nathan and D. H. Bodycomb, M.C., Captains (Honorary Majors) C. F. MacDonald and G. C. Scantlebury and Captains M. B. O'Sullivan, F. R. Meagher and N. F. Freemantle. The following officers are retired, 4th May, 1950: Honorary Majors N. T. Bull, J. H. Shaw and F. J. Colahan and Honorary Captain A. N. Dickson.

4th Military District.—The following officers are placed upon the Retired List (4th Military District) with permission to retain their rank and wear the prescribed uniform, 17th May, 1950: Lieutenant-Colonel (Honorary Colonel) F. L. Wall, M.C., Lieutenant-Colonels R. L. Kenihan, M.C., and E. B. Jones, Major (Honorary Lieutenant-Colonel) G. H. B. Black, Majors J. R. S. G. Beard, M.C., W. H. Collins, D.S.O., W. L. Smith, M.C., J. C. Mayo, L. G. Tassie, D.S.O., J. W. Flood, W. J. W. Close, K. N. Steele, A. H. Guymer and J. G. Sleeman, Captain (Honorary Major) L. W. Linn, Captains A. C. Wilton, J. G. Sweeney, P. W. Rice, R. A. Haste, G. H. Burnell, G. S. Shipway, A. K. Gault, E. L. Symons and G. Wien-Smith, and Honorary Captains S. C. M. Downing, R. J. Sargent and J. D. Mill. The following officers are retired, 17th May, 1950: Honorary Captains H. M. J. Halloran, K. McEwin, G. H. Kendrew, J. A. Rolland, W. Hamilton, N. R. Bennett, L. L. Davey and O. M. Moulden.

5th Military District.—The following officers are placed upon the Retired List (5th Military District) with permission to retain their rank and wear the prescribed uniform, 10th May, 1950: Major O. R. Corr and Captain (Honorary Major) A. A. Hill. Honorary Captain A. E. Williams is retired, 10th May, 1950. To be Honorary Captain, 2nd April, 1950: Clement Vernon Love.

ROYAL AUSTRALIAN AIR FORCE.

Reserve: Medical Branch.

Rollo Greenless (281231) is reappointed to a commission with the temporary rank of Squadron Leader, 3rd May, 1950.

The following are appointed to commissions with the rank of Flight Lieutenant: Kenneth Barnden Brown (297510); Peter Augustine Rogers (267886), 5th May, 1950; Roderick Ronald Collmann (450366), 15th May, 1950.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1939, of New South Wales, as duly qualified medical practitioners:

Behrndt, Arthur Clive, M.B., B.S., 1948 (Univ. Adelaide), 61 James Street, Punchbowl.

Jackson, Edward Ashley, M.B., B.Ch., 1948 (Univ. Dublin), 114 Birriga Road, Bellevue Hill.

Johnston, Edward Douglas, M.B., B.S., 1946 (Univ. Queensland), Royal Alexandra Hospital for Children, Camperdown.

Pendrill, Alice Mary, M.R.C.S. (England), L.R.C.P. (London), 1946, 5 Dulwich Road, Chatswood.

The following additional qualifications have been registered:

Horn, Craig Angus, 463 Peel Street, Tamworth (M.B., B.S., 1942, Univ. Queensland), D.D.R., 1949 (Univ. Sydney).

Howarth, Vernon Stuart, 20 Hodgson Avenue, Cremorne (M.B., B.S., 1939, Univ. Sydney), M.S. (Univ. Sydney), F.R.A.C.S., 1950.

Lang, William Robson, Penshurst Street, Willoughby (M.B., B.S., 1945, Univ. Sydney), F.R.C.S. (England), 1949.

Ritchie, Harold John, 225 Macquarie Street, Sydney (M.B., Ch.M., 1908, Univ. Sydney, F.R.A.C.P., 1939), F.R.C.P. (London), 1950.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts*, 1939 *to* 1948 of Queensland, as duly qualified medical practitioners:

Thatcher, Alfred Ronald, L.R.C.P. (London), M.R.C.S. (England), 1942, 96 Constitution Road, Windsor, Brisbane.

Condon, Robert Francis, M.B., B.S., 1946 (Univ. Adelaide), c.o. District Hospital, Collinsville.

Johnson, Alexander, M.B., B.Ch., B.A.O., 1939 (Queen's Univ., Belfast), c.o. Hospitals Board, Toowoomba.

Morton, Max Robson, M.B., B.S., 1947 (Univ. Sydney), c.o. Flying Doctor Service, Cloncurry.

O'Reilly, Esther Isla, M.B., B.S., 1944 (Univ. Sydney), 25 Pembroke Street, Cairns.

The following additional qualifications have been registered:

Gallagher, Michael Joseph (junior), 149 Wickham Terrace, Brisbane, M.Ch.Ortho. (Univ. Liverpool), 1948.

McSweeny, Anthony Francis, Wickham House, Wickham Terrace, Brisbane, F.R.A.C.S., 1950.

TASMANIA.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners:

Archer, Geraldine, M.B., B.S., 1948 (Univ. Adelaide), Launceston General Hospital, Launceston.

Cocks, Alan Malcolm Vawdry, M.B., B.S.[1] (Univ. Adelaide), Department of Health, Tasmania.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Week-End Course at Taree.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that a post-graduate week-end course will be held at the Municipal Council Chambers, Taree, in conjunction with the Eastern District Medical Association, on Saturday and Sunday, August 19 and 20, 1950. The programme will be as follows.

Saturday, August 19.—2.15 p.m., registration; 2.30 p.m., "Acute Abdominal Conditions", Dr. Kenneth W. Starr; 3.45 p.m., "Various Patterns of Rheumatoid Arthritis", Dr. L. J. Parr; 4.45 p.m., discussion; 5 p.m., annual general meeting of the Eastern District Medical Association.

Sunday, August 20.—9 a.m., annual general meeting of the Eastern District Medical Association; 9.30 a.m., "Anæmias", Dr. Kenneth T. Hughes; 10.45 a.m., "Ankylosing Spondylitis", Dr. L. J. Parr; 1.30 p.m., "Recent Advances in Surgery", Dr. Kenneth W. Starr; 2.30 p.m., "Cardio-Vascular Diseases", Dr. Kenneth T. Hughes.

[1] The year of graduation is not stated in the *Tasmanian Government Gazette*, from which this information is taken.

The fee for the course will be £2 2s. Those wishing to attend are requested to notify Dr. Angus McNeil, Honorary Secretary, Eastern District Medical Association, River Street, West Kempsey, as soon as possible.

Week-End Course in Pulmonary Tuberculosis.

The Post-Graduate Committee in Medicine in the University of Sydney announces that a week-end course in pulmonary tuberculosis will be held on Saturday and Sunday, September 9 and 10, 1950, under the supervision of Dr. Cotter Harvey. The course will be held at the Royal North Shore Hospital, Repatriation General Hospital and Royal Prince Alfred Hospital, and will consist of the following lectures.

Saturday, September 9.—2 p.m., "Diagnostic Problems in Pulmonary Tuberculosis", Dr. C. G. Bayliss; 2.30 p.m., "The Use and Abuse of Rest in Pulmonary Tuberculosis", Dr. Bruce White; 3 p.m., "Symptomatic Treatment", Dr. Cotter Harvey; 4 p.m., "Returning the Patient to Normal Living", Dr. R. H. Brent; 4.30 p.m., "Tuberculous Empyema", Dr. Ian Monk.

Sunday, September 10.—10 a.m., "Tuberculous Laryngitis", Dr. R. H. Bettington; 10.30 a.m., "Common Complications of Pulmonary Tuberculosis", Dr. A. W. Morrow; 11.30 a.m., "Resection in Pulmonary Tuberculosis", Dr. M. P. Susman; 12 noon, radiological demonstration, Dr. J. Colclough; 2.15 p.m., "Epidemiology and Control of Pulmonary Tuberculosis", Dr. Cotter Harvey; 2.45 p.m., "The Management of Progressive Pulmonary Tuberculosis", Dr. H. Maynard Rennie; 3.15 p.m., "Evaluation of Activity in Pulmonary Tuberculosis", Dr. Maurice R. Joseph; 4.15 p.m., clinico-pathological conference, Dr. Edgar Thomson, Dr. V. J. McGovern and staff of the thoracic unit of the Royal Prince Alfred Hospital.

Fee for attendance will be £2 2s. Those wishing to enrol are requested to apply, as soon as possible, to the Course Secretary, the Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney, from whom programmes may be obtained. Telephones: BU 5238, BW 7483.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED JULY 15, 1950.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.	Australia.[3]
Ankylostomiasis	*	..	..	..	..	..	..	..	..
Anthrax	*	..	..	..	..	..	..	..	..
Beriberi	*	*	*	*	*	*	..	*	..
Bilharziasis	*	..	..	..	..	..	..	..	..
Cerebro-spinal Meningitis	2(1)	4(2)	..	1	4(2)	..	..	..	11
Cholera	..	..	..	..	..	..	..	..	..
Coastal Fever(*a*)	*	*	*	*	*	*	*	*	..
Dengue	*	*	*	*	*	*	..	*	..
Diarrhœa (Infantile)	..	..	6(6)	..	..	..	..	..	6
Diphtheria	15(3)	4(2)	11(9)	1	4(3)	..	..	..	35
Dysentery (Amœbic)	*	..	1(1)	..	..	..	..	..	1
Dysentery (Bacillary)	*	3(2)	..	..	*	..	..	..	3
Encephalitis Lethargica	..	..	..	..	..	..	..	..	..
Erysipelas	*	*	*	..	*	*	..	..	..
Filariasis	*	..	..	..	..	..	..	..	..
Helminthiasis	*	..	*	*	*	*	*	*	..
Hydatid	*	..	*	*	..	*	*	..	..
Influenza	*	*	*	..	*	*	..	*	..
Lead Poisoning	*	*	..	*	*	*	*	*	..
Leprosy	..	..	..	..	..	..	..	..	..
Malaria(*b*)	*	..	..	..	..	..	..	..	..
Measles	*	*	*	171(53)	*	..	..	3	174
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	7(2)	1(1)	2	12(10)	4(2)	..	..	1	27
Psittacosis	*	..	*	..	*	*	*	*	..
Puerperal Fever	..	..	1	..	..	..	..	..	1
Rubella(*c*)	*	*	..	..	..	*	..	1	1
Scarlet Fever	20(10)	20(10)	3(2)	13(9)	3(1)	2(1)	..	..	61
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	*	..	..	*	*	*	*	*	..
Trachoma	*	..	..	*	*	*	*	*	..
Tuberculosis(*d*)	23(18)	16(8)	18(9)	9(7)	18(14)	5(4)	..	..	89
Typhoid Fever(*e*)	..	..	..	..	..	..	..	..	..
Typhus (Endemic)(*f*)	1	..	..	..	..	..	..	..	1
Undulant Fever	..	..	..	..	..	..	..	..	..
Weil's Disease(*g*)	*	*	38	*	*	*	*	*	38
Whooping Cough	*	*	*	3(3)	*	*	..	..	3
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] The form of this table is taken from the *Official Year Book of the Commonwealth of Australia*, Number 37, 1946–1947. Figures in parentheses are those for the metropolitan area.
[2] Figures not available.
[3] Figures incomplete owing to absence of returns from the Northern Territory.
* Not notifiable.
(*a*) Includes Mossman and Sarina fevers. (*b*) Mainly relapses among servicemen infected overseas. (*c*) Notifiable disease in Queensland in females aged over fourteen years. (*d*) Includes all forms. (*e*) Includes enteric fever, paratyphoid fevers and other Salmonella infections. (*f*) Includes scrub, murine and tick typhus. (*g*) Includes leptospiroses, Weil's and para-Weil's disease.

SEMINARS AT ROYAL PRINCE ALFRED HOSPITAL.

THE following seminars will be held at Royal Prince Alfred Hospital, Camperdown, New South Wales, during the second half of 1950. Each seminar is held on Friday from 1.15 to 2.15 p.m. in the A2 lecture theatre. All medical graduates are invited to attend.

August 11: Endocrinology and Metabolism Section, "Modern Views on Anuria".

August 18: Hæmatology Section, "Marrow Examination in Hæmatology".

August 25: Neurology Section, "Reconsideration of Trigeminal Neuralgia" (Dr. G. Phillips).

September 1: No seminar.

September 8: Gastro-Enterology Section, "Gastritis" (Dr. R. Doig, Melbourne).

September 15: Pædiatric Section, "Respiratory Obstruction in Infancy".

September 22: Thoracic Section, "Lung Abscess".

September 29: Pathology Section, clinico-pathological conference.

October 6: Cardio-Vascular Section, "*Cor Pulmonale* I".

October 13: Cardio-Vascular Section, "*Cor Pulmonale* II".

October 20: Endocrinology and Metabolism Section, "Diabetic Coma".

October 27: Gastro-Enterology Section, Journal Club.

November 3: Neurology Section, Journal Club.

Royal Australasian College of Surgeons.

COURSE FOR PRIMARY F.R.A.C.S. EXAMINATION.

A COURSE will be conducted in Sydney suitable for candidates for the primary F.R.A.C.S. examination. It will commence on August 14, 1950, and will last for approximately three months. The fee will be thirty guineas. Those desirous of taking the course are requested to hand in their names to the Secretary of the Post-Graduate Committee in Medicine in the University of Sydney, 131 Macquarie Street, Sydney, who will be arranging the course.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Martin, Kelvin Sylvester, M.B., B.S., 1950 (Univ. Sydney), Western Suburbs Hospital, Croydon.

Black, Margaret, M.B., B.S., 1939 (Univ. Sydney), 167 John Street, Singleton, New South Wales.

Black, Michael James Morrison, M.B., 1941 (Univ. Sydney), 167 John Street, Singleton, New South Wales.

White, Dennis Howard, M.B., 1950 (Univ. Sydney), 22 Woodward Avenue, Strathfield.

Webster, Zena Barber, M.B., B.S., 1948 (Univ. Sydney), 21 Ainslie Street, Kingsford.

Maybloom, Bernard Laurence, M.B., B.S., 1950 (Univ. Sydney), 52 Palmer Street, Rose Bay North.

Medical Appointments.

Dr. P. P. Bateman and Dr. Merna A. Muller have been appointed associate medical officers at the Royal Adelaide Hospital, Adelaide.

Dr. John Orde Poynton has been appointed director of the Institute of Medical and Veterinary Science, South Australia.

Dr. J. G. Linn and Dr. B. R. Walsh have been appointed associate medical officers at the Royal Adelaide Hospital, Adelaide.

Notice.

FAIRFAX READING MEMORIAL PRIZE.

THE Dental Alumni Society of the University of Sydney advises that the Fairfax Reading Memorial Prize for 1950 has been awarded to Dr. A. C. Gabriel for his paper "Genetic Types in Teeth".

Diary for the Month.

AUG. 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
AUG. 11.—Queensland Branch, B.M.A.: Council Meeting.
AUG. 14.—Victorian Branch, B.M.A.: Finance, House and Library Subcommittee.
AUG. 15.—New South Wales Branch, B.M.A.: Medical Politics Committee.
AUG. 16.—Western Australian Branch, B.M.A.: General Meeting.
AUG. 17.—Victorian Branch, B.M.A.: Executive Meeting.
AUG. 22.—New South Wales Branch, B.M.A.: Ethics Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney)—All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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